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William Macewen

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The Journal of Bone and Joint Surgery

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OF
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American Orthopaedic Association
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We are also anxious to report the Proceedings of Colleges, Universities, Societies and Associations in every part of the world in so far as they contribute to the advance of orthopaedic surgery. A measure of international co-operation in publication has already been achieved and this will be fostered and, we hope, increased.

Manuscripts offered for publication in the British volume should be addressed to the Editor, Journal of Bone and Joint Surgery, 82 Portland Place, London. The Editorial Secretary of the Board is at the service of authors, and will assist in any way she can. Manuscripts offered for publication in the American volume should be addressed to the Editor, Journal of Bone and Joint Surgery, 8 The Fenway, Boston 15, Massachusetts.

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GALLIE, W. E. (1948): Recurrent Dislocation of the Shoulder. Journal of Bone and Joint Surgery, **30 B**, 6.

The names of Journals should be given in full; the "World List" of abbreviations is not used. Every bibliography and list of references will be checked by the Editor, but much delay will be avoided if authors would be good enough to pay careful attention to accuracy.

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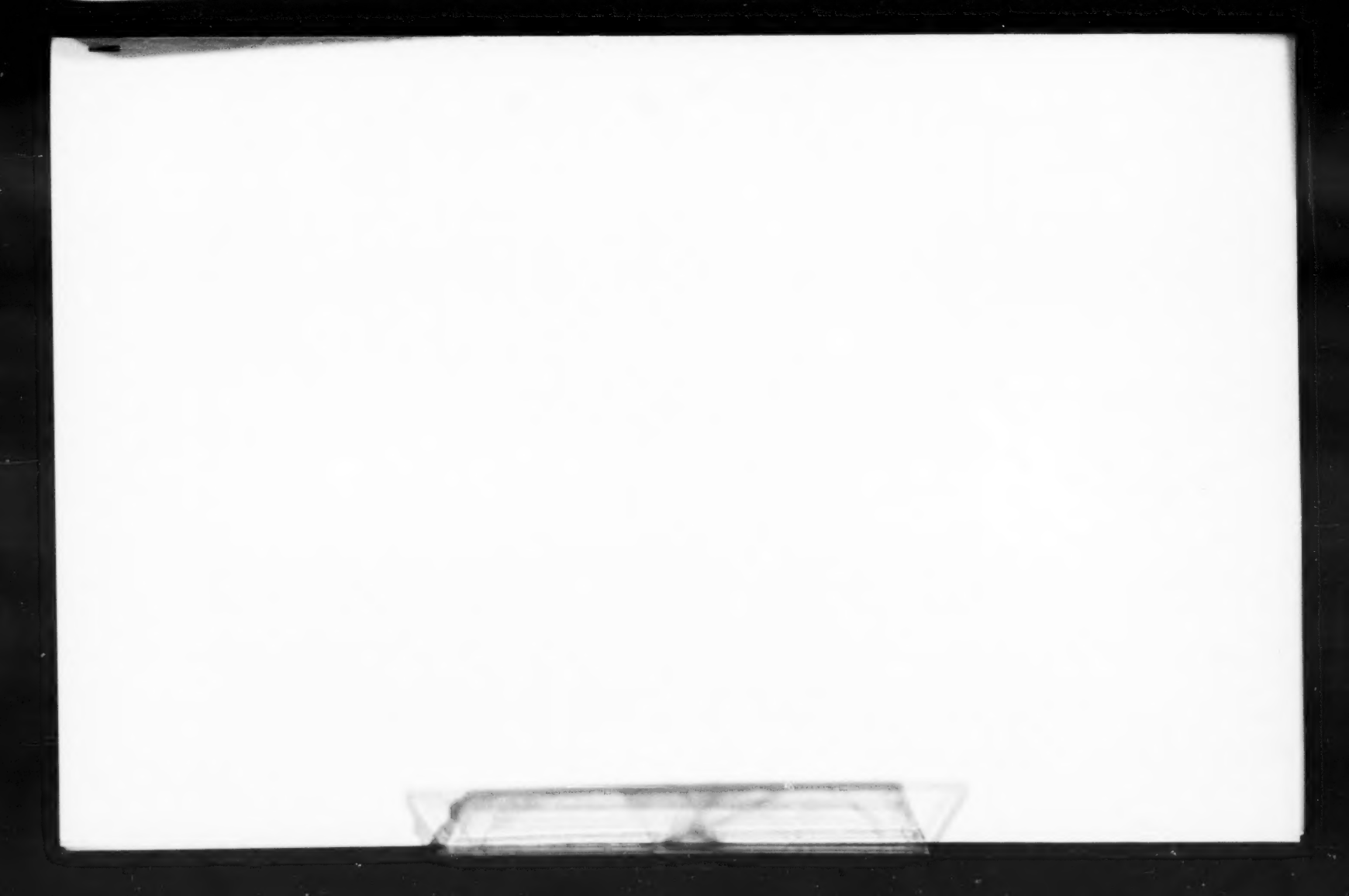

The Journal of Bone and Joint Surgery

6th February 1952

We have learned with deep regret of the death of His Majesty the King. His reign was respected and admired throughout the world. One small facet was the support that His late Majesty gave to this Journal of the British Commonwealth of Nations and the United States of America. A letter was at once sent to the Queen Mother, Her Majesty Queen Elizabeth:

> With humble duty and sustained sympathy Your Majesty's most obedient servants the Fellows and Members of the British Orthopaedic Association beg to express their sense of very deep loss of a King who reigned with such understanding, their condolence in Your Majesty's personal distress, and their profound admiration for the courage, equanimity and warm-hearted kindness that Your Majesty has displayed in recent months and years.

A few hours later we received a cablegram from Dr William Rogers, Editor-in Chief, and the American Editorial Board:

> To our British colleagues with the deep regrets and sincere sympathy of the American Board of Editors in the passing of your beloved King.
>
> WILLIAM ROGERS.

Later, after the proclamation of accession of Her Majesty the Queen, a telegram was sent to Buckingham Palace:

> Orthopaedic surgeons of Britain, Canada, Australia, New Zealand and South Africa with enduring loyalty to their Queen are joined by orthopaedic surgeons of the United States of America in deep and compassionate sympathy and with sure and confident trust in the future.

We have received this telegram in reply from Her Majesty's Private Secretary:

> To Sir Reginald Watson-Jones.
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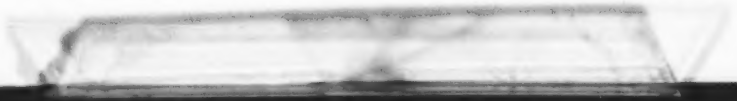

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The Journal of
Bone and Joint Surgery

EDITORIALS

JOINT MEETING OF THE ORTHOPAEDIC SURGEONS OF THE ENGLISH-SPEAKING WORLD, 1952

After the first world war, the first joint meeting of the American and British Orthopaedic Associations was held in London under the presidency of Robert Jones. It was attended by Albee, Goldthwait, Royal Whitman, Arthur Keith, Hey Groves, Elmslie, Bankart, Laming-Evans, Fairbank, Platt, Girdlestone, McMurray, Naughton-Dunn, Steindler, Smith-Petersen, Winnett-Orr, Putti and others. It was a meeting of the giants of orthopaedic surgery.

Soon after the second world war there was a joint meeting of the American, British and Canadian Orthopaedic Associations in Quebec under the presidencies of Harris of Toronto, Malkin of Nottingham and Samson of Montreal. It was attended by the leaders—but a feature of the meeting was the contribution of young surgeons who have since formed the A.B.C. club of America, Britain and Canada: Hamilton Allan, Donald Blanche, Verne Inman, John Fahey, Benny Obletz, Carroll Larson, William Bickel, Hugh Smith, Ramsey Straub and Ben Fowler of the United States; Ted Dewar, Leo Walker, Frank Paterson, William McKinnon and Roger Gariepy of Canada; John Adams, John Charnley, Fred Durbin, James Ellis, John Fairbank, Cecil Langton, Philip Newman, James Patrick, Marion Pearson, Ian Smillie, Edgar Somerville, Derek Strange and James Wishart of Great Britain. We speak of Verne, Carroll and Ted—but they are now professors of orthopaedic surgery; we speak of John, Hugh, Fred and the rest—but they are now the leaders or deputy-leaders of orthopaedic surgery in their cities. This was indeed a meeting of the coming giants of orthopaedic surgery.

What of the third joint meeting? It will be held in London in the first week of July 1952—a joint meeting of orthopaedic surgeons of the English-speaking world. There will be official representation of the Orthopaedic Associations of America, Britain, Canada, Australia, New Zealand and South Africa with other surgeons from the British Commonwealth; and we shall have guests from Europe and South America who speak not only their own language but ours as well. Can we expect that giants, coming giants or future giants will be found in this meeting? If not, we shall have failed.

This is the first time in history that all orthopaedic surgeons of the English-speaking world have planned to hold their annual meetings at the same moment, and to meet in London. The occasion is of historic importance and Her Majesty the Queen hopes to attend the Inaugural Ceremony and present each visiting president with a jewel, designed by a leading member of the Worshipful Company of Goldsmiths, and given by the fellows and members of the British

Reginald Watson-Jones
President of the British Orthopaedic Association.

Orthopaedic Association. We trust that these presidential badges of office will be worn in perpetuity at all future meetings of the American, Canadian, Australian, New Zealand and South African Orthopaedic Associations as an emblem of the warm friendship and close esteem with which our friends in these Associations are held in Great Britain. The ceremony will be in the University of London on the morning of Monday, June 30, attended in academic robes and with the insignia of office by the Chancellor and the Principal of the University of London and the Presidents of the several Royal Colleges of Surgeons and of Physicians of Great Britain.

The scientific meetings and discussions will be held in the Beveridge Hall of the University of London each morning of the week, and there will be clinical demonstrations on two afternoons at various London hospitals. A Robert Jones Memorial Lecture and a Moynihan Lecture will be delivered in the Royal College of Surgeons of England in Lincoln's Inn Fields. Some of the treasures of the College and the Hunterian Museum will be on display. There will be a continuous showing of cinematograph films throughout the meeting.

Immediately after the official programme in London a series of visits is being arranged to all orthopaedic centres throughout the country. We hope that visiting surgeons will stay for a long time, and in order to help in planning their tours a detailed list of routine activities of all orthopaedic clinics in this country is printed in this issue of the Journal. Many believe that the greatest benefit is gained if a few visiting surgeons spend a week or more in one clinic, and every surgeon in Great Britain whose routine activity is detailed in this issue of the Journal will be more than ready to welcome visitors. For those whose stay must be curtailed, a few lightning tours will be planned—for example: Oswestry, Liverpool, Manchester and Edinburgh; Norwich, Cambridge, Nottingham and Edinburgh; Exeter, Bath, Bristol, Stratford-upon-Avon and Oxford or Birmingham; and there will be others. Every clinic will be open to every visitor for as long as he cares to stay.

Throughout the week of the official meeting there will be evening social engagements for fellows and members with wives and families, including a reception at Claridges on June 29 when we hope that visitors and guests will complete their plans for personal entertainment; a Ballet performance at the Royal Opera House, Covent Garden; an official dinner at the Dorchester Hotel when the toasts will be the Guests, Orthopaedic Surgery, and the English Language; and an Independence Day Celebration at Hurlingham Club on July 4 with dancing and pyrotechnics. There will be dinners of the Robert Jones Clubs of London and New York, of the old Oswestrians, old M.Ch.Orthians, the A.B.C. Club, and the combined Editorial Boards of this Journal.

But we are still concerned about the entertainment of wives and families of surgeons, and of the surgeons themselves when they have had a surfeit of academic discussion. They can go to Wimbledon or Henley; to the Tower of London, Westminster Abbey or the Houses of Parliament; to Windsor or St Albans; to Cambridge or Oxford; to the Painted Hall of the Royal Naval College, the Thames and the London Docks; to Stratford-upon-Avon and the Shakespeare Theatre; to Cowdray Park to see polo; or to Sunningdale or Walton Heath to play golf. There is so much that can be done. On this there are two schools of thought. Some think that tours should be planned and rigidly organised; others believe that, having been informed, each visitor should choose and plan for himself. At all events it is a free country and everyone can decide with independence. We will try to satisfy each school of thought.

Will every visiting surgeon and his wife tell us what they would enjoy most? Will every member of the British Orthopaedic Association find out for themselves what our visitors would like—and arrange it in private parties?

In this issue of the Journal we reproduce photographs of the presidents of the British, American and Canadian Orthopaedic Associations. In the next issue there will be photographs of the presidents of the Australian, New Zealand and South African Orthopaedic Associations.

FREMONT A. CHANDLER
President of the American Orthopaedic Association

R. Graham Huckell

President of the Canadian Orthopaedic Association.

TRAUMATIC CHYLOTHORAX

In this issue Wright and Gardner describe a case of traumatic chylothorax following complete dislocation of the thoracic spine; the severity of the injury prevented diagnosis before death, but their paper serves to remind us of many interesting facts about this rare condition, not least the dramatic fall in mortality produced by modern treatment. The condition was first described by Bartolet in 1633. The number of fully authenticated cases had risen to only fifty-eight by 1946, with an overall mortality of 50 per cent (Loe). In that year Lampson showed that traumatic chylothorax could be cured by ligation of the thoracic duct in the chest; Meade *et al.* (1950) confirmed the practicability of direct surgical attack, but emphasised that the simpler method of closed drainage of the chest had effected a cure in their last four cases. Nine of the last eleven cases reported have been cured by closed drainage or ligation of the thoracic duct. Excellent reviews have been published by Shackelford and Fisher (1938) and Meade *et al.* (1950). It is interesting to note that many of the recorded cases arose from trivial injuries hardly requiring medical attention—falling down stairs, "back diving," paroxysms of coughing; in quite a few cases trauma to the duct occurred during thoracic operations.

In a typical case there is an interval of two to seven days after injury before the chylothorax begins to form; rarely this latent period may be as long as four months. When the associated injury is sufficiently severe, slow recovery from shock may explain this delay, as suggested by Wright and Gardner in this issue. When the injury is trivial, it is assumed that the intact mediastinal pleura contains the chyle until increasing pressure enables it to find a way into the pleural cavity. Once started, the chylothorax increases rapidly. The whole of the normal output of chyle (two litres a day) has often been aspirated from the pleura. Undiagnosed, the chylothorax rapidly produces death by asphyxia; more usually, the dramatic relief of symptoms produced by aspiration, and the enormous quantities of thin milky fluid (not unlike pus) requiring removal daily from the pleural cavity of a patient with a normal temperature, should suggest the diagnosis, when pathological confirmation will be quickly forthcoming. The marked fall in circulating lymphocytes that necessarily accompanies chylothorax may aid the diagnosis. Once the diagnosis is established, treatment should follow the lines suggested by Meade. The patient should be placed on a low-fat, high-protein diet, with an adequate fluid content, and the pleura emptied daily by aspiration. If the chylothorax persists unabated after ten days of this treatment, closed drainage with continuous suction should be established. Delay is dangerous, both because the long continued loss of protein and fat will eventually lead to death by inanition and because the danger of encapsulation of the collapsed lung is real. Chyle is irritant to the pleura, and a fibrous capsule may form that will require removal by a tedious decortication before expansion of the lung can take place.

If closed drainage is a failure, then the thoracic duct should be ligated just above the diaphragm; a portion should always be removed for histological confirmation. Right thoracotomy gives the better exposure and should be used unless decortication of the left lung appears necessary. Simple aspiration has in the past cured 50 per cent of traumatic chylothoraces; Meade's experience with closed drainage is much more encouraging and, properly performed, ligation of the duct should cure all the rest. There is abundant evidence, both clinical and experimental, that ligation of the thoracic duct is compatible with good health. The intravenous administration of chyle aspirated from the chest has been used since 1908 to minimise the loss of protein and fat; Blalock and his collaborators (1942) successfully administered forty-two litres of chyle in this way. Two deaths have occurred during this procedure, however, and it is perhaps best avoided.

G. C. Laurie Pile.

SPONTANEOUS FRACTURES OF BOTH FIRST RIBS

S. Aubrey Jenkins, Eastbourne, England

Formerly Lecturer in Orthopaedic Surgery, Postgraduate Medical School, London

This fracture has been variously reported as a developmental anomaly, a synchondrosis or a pseudarthrosis, but most authors now regard it as a true fracture. As far as can be ascertained, only 263 cases of isolated fracture of the first rib have been recorded in the literature and only seventeen of these were bilateral. The majority of the recorded cases have been found incidentally during routine radiography of young healthy adults, notably members of the Services (Alderson 1944 and 1947, Etter 1944, Sjögren 1942). Most of the patients have been unable to remember any injury or any symptoms attributable to a fracture, although a few recollect a sudden pain in the neck and shoulder when lifting or straining, followed by discomfort which persists for a few days afterwards. The fracture is usually symptomless at the time of radiography. Only fifteen cases have been reported with clinical symptoms sufficiently severe for an early radiograph to be taken and even in some of these patients the fracture appears to have been unsuspected before that examination.

When the fracture is first diagnosed, the radiographs may show either a simple linear crack in the rib or a pseudarthrosis with widened, irregular, sclerotic opposing surfaces, or the fracture may already be soundly healed with varying degrees of residual thickening of the rib about the fracture site. Alderson (1947) has shown by serial radiographs that non-union or a false joint often follows this fracture although bony union does occur in some. Symptomless established non-union is often found (Hartley 1945). The fracture is almost confined to males, usually under twenty years of age.

ANATOMY AND ETIOLOGY

The first rib is short, broad and flat. The scalenus anterior and scalenus medius muscles are inserted into its upper surface on either side of the groove for the subclavian artery. The subclavian vein lies on a second, shallower, groove in front of the scalenus anterior. Sudden contraction of the scalenus anterior or medius would tend to pull the rib upwards against the resistance of its anterior and posterior attachments. The intercostal muscles are attached to the convex outer margin of the first rib along the greater part of its length. Anterior to the scalene muscles, the rib also gives attachment to the first digitation of the serratus anterior, the subclavius muscle and the costo-clavicular ligament. It has been suggested that these attachments render the anterior half of the rib less mobile and that sudden powerful contraction of the scalene muscles may fracture the rib at its thinnest segment—where the subclavian artery crosses it.

Some deny that this lesion is a fracture at all and attribute it to a developmental anomaly, because of the absence of a history of injury and the rarity of non-union of fractures in other ribs (Bowie and Jacobson 1945, Sycamore 1944). The body of the first rib may arise from two centres of ossification, and the junction may persist as a synchondrosis (Gershon-Cohen and Delbridge 1945) but this lies farther back than the usual site of a fracture and the line of division between the two bony segments lies in a sagittal plane and not transversely as in a fracture.

The sex incidence is strongly in favour of an acquired lesion, and so is the history of recent unaccustomed physical effort in some of the patients. In a few cases an earlier radiograph has been available showing a previously normal rib (Alderson 1947). It is known that fractures of other ribs, especially the seventh and eighth, are sometimes caused by sudden muscular

effort (Kleiner 1924, British Medical Journal 1949) or by severe or constant coughing (Cohen 1949, Palfrey 1924, Paulley *et al.* 1949). In the lower ribs such fractures are usually situated at a point where two muscle masses pull in different directions. A simple muscle strain can also produce a fracture of the first rib (Aitken and Lincoln 1939), and the most frequent cause, when known, appears to be unaccustomed exertion and especially heavy lifting-strains (Garber 1944). Coughing alone does not appear to have precipitated a fracture of the first rib.

Direct external violence seldom fractures the first rib, which is deeply situated and protected by the shoulder girdle. However, the first recorded isolated fracture of the first rib was due to repeated kicks during a drunken brawl and led to rapid death from rupture of the subclavian vessels (Jones 1869). A fracture of the first rib may occasionally be associated with adjacent fractures of the clavicle, scapula, spine or lower ribs (Knoepp 1941) and such direct injuries are often complicated by injury to the subclavian vessels, the brachial plexus or the pleura (Breslin 1937).

Indirect violence causing a fracture of the first rib has been described by Powell (1950). One of his patients collided shoulder to shoulder with another player at football, another fell on to his outstretched hand and a third suffered forcible hyperabduction of the arm. In all three cases there was immediate pain, and the radiograph showed a recent fracture. Sudden forcible flexion of the neck to one side may also fracture the first rib, presumably from tension of the scalene muscles (Aitken and Lincoln 1939).

It seems likely that this fracture may sometimes be unrelated either to muscular pull or to injury and is then comparable with the stress or fatigue fractures occurring in the metatarsals, tibia, fibula and neck of femur (Hartley 1945). Proctor *et al.* (1945) have reported three cases in which the fracture was caused by the carrying of heavy weights slung from the shoulder.

CLINICAL FEATURES

In the few patients who present themselves with symptoms immediately after the fracture, the clinical findings are characteristic if the possibility of this fracture is kept in mind. The main feature is pain, which usually begins acutely and causes the patient to support the arm with his other hand as though he had sustained a fracture of the clavicle; but sometimes the pain develops only slowly. It persists as a vaguely localised pain at the base of the neck or in the upper chest, behind the clavicle or under the scapula. The pain is increased by abduction of the shoulder, and sudden unpremeditated movements of the arm in any direction may cause sudden stabs of pain. Occasionally, pain or paraesthesiae radiate down the arm (Aitken and Lincoln 1939) but always along its medial aspect, in the region supplied by the lower trunk of the brachial plexus. There may be weakness of the whole arm at first and the strength of the grip is diminished. This is directly related to the pain and rapidly recovers as the pain subsides.

There is no tenderness around the shoulder, which usually has a full range of movement. There may be a small area of acute accurately localised tenderness in the neck immediately over the fracture site; this lies behind the lateral border of the sternomastoid muscle at its insertion into the clavicle. The tenderness may not be present until several days after the fracture. There is no palpable swelling until callus has had time to form and this does not develop in less than a month. The mass of callus remains tender in the early stages and pressure over it may give referred pain down the medial side of the arm. Local compression of the chest does not provoke pain as it does in fractures of the other ribs, but simultaneous pressure over the clavicle and scapula may do so.

TREATMENT

No specific treatment is required. The pain or discomfort invariably subsides and the patient is usually free from symptoms within a few days irrespective of the treatment given,

and even though the fracture may not unite. A simple sling may be required and the patient should avoid heavy work or excessive movement of the arm until free from discomfort.

CASE REPORT

A healthy youth, aged sixteen years, was lifting a box weighing 70 lb. from the floor on to a high counter, when he suffered sudden pain behind the right clavicle, radiating into the shoulder and upper chest. The pain persisted as a dull ache with intermittent acute stabs when coughing, sneezing or deep breathing. The pain was increased by using the right arm and especially when lifting it to shave or to drink. On clinical examination two days later the pain was vaguely situated in the shoulder and behind the clavicle but could not be localised accurately and it did not radiate down the arm. Active movements of the arm and shoulder gave rise to acute twinges of pain, especially when any attempt was made to raise the arm above shoulder level. The range of passive shoulder movements was full and painless with the patient supine; but both active and passive movements increased the pain with the patient standing or sitting. No area of tenderness could be found despite a careful search in the neck and shoulder; and compression of the chest did not cause pain. Radiographs showed a transverse fracture of the first right rib in the region of the scalene tubercle. The fracture line was a mere crack and difficult to identify because it was obscured by the clavicle, but study of the rib contour showed a very slight lateral displacement of the posterior half of the rib in relation to the anterior half.

Progress—The symptoms were not sufficiently severe to require any form of immobilisation, but a course of short-wave diathermy was prescribed and the discomfort rapidly subsided. The patient was allowed to continue light work and instructed to avoid all heavy lifting.

A further radiograph was taken a week later, to include both first ribs. The fracture site in the right rib was more easily seen in this film and the left rib unexpectedly showed a large mass of callus with an irregular fracture line running transversely through it (Fig. 2). The fracture of the left rib was symptomless and the patient was unable to recall any incident, injury or symptoms relating to this region. A small, bony hard, painless swelling could be felt at the root of the neck on the left side, lying immediately behind the lateral margin of the sternomastoid muscle at its insertion into the clavicle. This was evidently the callus around the old fracture of the first left rib. No swelling could be felt on the right side.

In view of these clinical and radiographic findings a more detailed history was obtained. The patient had been working as a mattress maker for one and a half years—a dusty but not a heavy occupation. Two months before attending hospital he had developed a slight cough which persisted for three weeks and his family doctor sent him to a chest clinic for radiography. The films were subsequently obtained and show no abnormality of the first right rib, but the appearance of the left rib is identical with that in the more recent films (Fig. 1).

He changed his occupation and became a warehouse porter two months before the incident described, and it seems probable that the unaccustomed physical exertion was a factor contributing to the recent fracture of the right rib. On the other hand, the long-standing fracture of the left rib occurred some time before and without symptoms, which suggests that there was a predisposing weakness of the rib at this site.

When seen five weeks after the onset of symptoms the patient had only slight residual discomfort. Three weeks later still he was completely free from pain and was able to resume full heavy work as a porter. Now, for the first time, two months after the fracture, a small hard swelling could be felt at the fracture site in the first right rib in a position exactly corresponding with the mass on the left side. The swelling was slightly tender, and firm pressure on it produced a sharp pain radiating down the medial side of the arm and hand in the distribution of the lowest trunk of the brachial plexus. There were no objective signs of paresis, altered sensation or circulatory changes in the limb.

Further radiographs, with markers over the swellings, confirmed that the fracture on the

FIG. 1

Radiograph of the chest taken two and a half months before the onset of symptoms on the right side. It shows an old fracture of the first left rib with the fracture line still visible, but no lesion on the right side.

FIG. 2

Radiograph taken two weeks after the spontaneous fracture of the first right rib. The fracture is seen immediately above the upper margin of the clavicle. Note also the long-standing symptomless and previously unsuspected fracture of the first left rib, with considerable thickening of the bone at this point.

FIG. 3

Radiograph taken two months after the onset of pain on the right side. Metal markers have been placed directly over the swellings which were felt in the neck and these are seen to coincide with the fractures. Note mass of callus and pseudarthrosis at each fracture site.

right side had now developed a mass of callus, comparable to the enlargement of the left rib but with incomplete bony union. The markers could be seen lying directly over the fracture site and as near to them as the intervening soft tissue would permit (Fig. 3).

Final radiographs, taken four months after the onset of symptoms, showed a fully developed pseudarthrosis of each fracture site. The patient had continued without symptoms at full heavy work.

SUMMARY

1. A case of spontaneous fracture of a first rib is described.
2. Its evolution from a previously intact rib through the stage of a "linear" crack to pseudarthrosis has been traced.
3. An older pseudarthrosis was present on the opposite side.
4. Alderson's observation is confirmed—that the breach in the rib is acquired. There is no need to suppose a developmental anomaly of ossification, even in a bilateral case.

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THE INTERINNOMINO-ABDOMINAL OPERATION

Observations on a Series of Fifty Cases*

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The fifty patients on whom this paper is based were operated upon by surgeons of the Middlesex Hospital between 1922 and 1950. Thirty-three were men and seventeen were women. Their ages lay between fourteen and sixty; eight were under twenty and thirteen were fifty or over; the average age of the group was thirty-five.

INDICATIONS FOR AMPUTATION

Malignant disease was the indication for amputation in all cases except one, when the operation was performed for extensive persistent osteomyelitis (Table I). Amputation through the pelvic girdle was necessary in all cases because of the position of the lesion and because it offered the only reasonable hope of saving or prolonging life; in some cases there existed the need to relieve intolerable pain, or the limb had become a useless encumbrance; in some, local

TABLE I

Indications for Amputation

Neoplastic disease			49
Osteomyelitis			1
Tumours of bone		**Tumours of soft tissue**	
Chondrosarcoma	15	Spindle-cell sarcoma	10
Osteogenic sarcoma	5	Fibrosarcoma	9
Osteogenic sarcoma complicating Paget's disease	2	Round-cell sarcoma	2
Diffuse endothelioma (Ewing's tumour)	2	Myosarcoma	1
Osteoclastoma	1	Metastases in iliac glands secondary to carcinoma of vulva	1
Solitary angioendothelioma	1		
	26		23

resection and radiotherapy had been tried without success. Of twenty-six amputations for neoplasms of bone, eighteen were for tumours of the pelvic girdle and eight for tumours of the upper end of the femur. In the femur osteogenic sarcoma was the most frequent tumour and in the pelvic girdle the most usual was chondrosarcoma—the neoplasm most often encountered in this series. Of the soft-tissue tumours nineteen were spindle-cell sarcomata or fibrosarcomata, and most of these were distal to the inguinal ligament.

* Sixty-three operations of this nature have been carried out by surgeons of the Middlesex Hospital up to date.

The case of osteomyelitis was unusual. The patient was a woman who had repeated acute attacks of osteomyelitis of the lower end of the femur and the ischium, in one of which she developed a metastatic abscess of the left iliac fossa around the external iliac vessels. This led to thrombosis and gangrene of the foot, making amputation necessary. She survived for only two weeks after operation and probably died from sulphonamide anuria, a condition imperfectly recognised at that time. The case in which radical amputation was undertaken in the later stages of carcinoma of the vulva is also interesting. The primary growth had been excised three years earlier, and interinnomino-abdominal amputation alone seemed to promise

FIG. 1

Chondrosarcoma of the upper end of the femur. The patient was alive and well three years after amputation had been performed.

adequate removal of the metastases in the inguinal and iliac glands; moreover, the leg had become grossly oedematous and painful as a result of the secondary deposits and treatment by radiotherapy. The patient is now alive and well, six years after operation. Amputation was undertaken for two cases of diffuse endothelioma of bone (Ewing's tumour) affecting the ischiopubic rami and the ilium respectively. The first was among the earlier amputations, and the patient died from shock; the second patient underwent successful amputation in 1949, but a year later developed a local recurrence which responded satisfactorily to deep X-ray therapy. Such tumours are probably best treated by irradiation. Amputation for an osteoclastoma was the second case in this series and the first patient to survive the operation. The nature of the tumour was not recognised before operation, and amputation

was carried out because of the degree of involvement of the innominate bone. The man still leads a useful life, twenty-two years after operation. The case of solitary angio-endothelioma affecting the ilium has already been reported in this journal (Gordon-Taylor and Wiles 1949). These tumours are uncommon, but like other tumours of vascular origin in bone they may respond to irradiation. No certain pre-operative diagnosis could be made in this case, however, since an attempt at biopsy had to be abandoned because of severe haemorrhage. The lesion presented as a highly vascular malignant tumour eroding bone, without evidence of a primary growth elsewhere, and amputation therefore seemed clearly indicated.

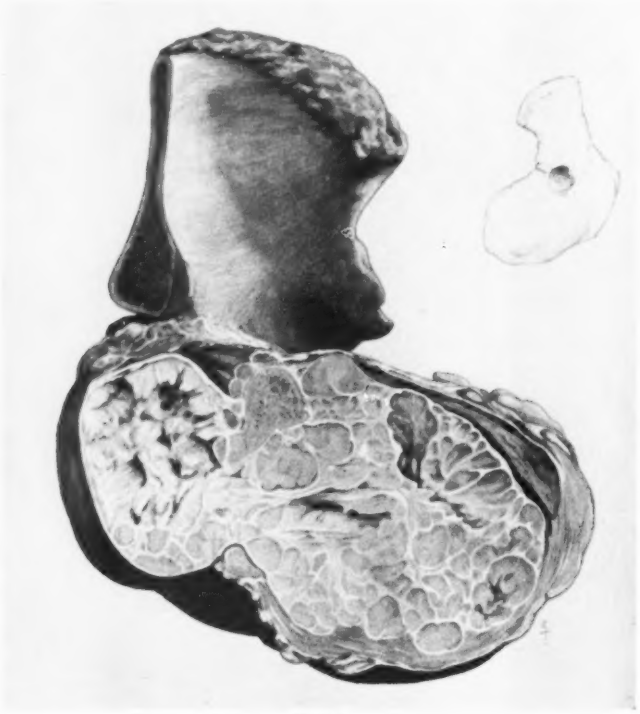

FIG. 2

Chondrosarcoma of the ischiopubic region of the left hip bone infiltrating the prostate. Hindquarter amputation and prostatectomy were undertaken; the continuity of the urethra was restored by suturing the membranous urethra to the neck of the bladder. The patient made a good recovery from the operation.

TECHNIQUE OF OPERATION

Penicillin and streptomycin therapy is started the day before operation, and ten pints of matched blood are held in readiness. The anaesthetic of choice is thiopentone, gas and oxygen. The patient is placed on the operating table lying on the side opposite to the lesion, and is turned slightly on to his back. The position is maintained by a support against the lower ribs and thoracic spine, well clear of the iliac crest, and by an assistant who stands at the far end of the operating table on the same side as the surgeon and holds the limb. The uppermost arm is supported on a rest of the type used during nephrectomy, and transfusions

are given into the veins of this limb. A sheet of oiled silk is stitched over the anus and a catheter is passed—formerly this was tied in, but a Foley catheter is now employed. An Esmarch bandage is then applied to the affected limb.

The usual incision is in two parts, anterior and posterior. The anterior, which is made first, lies immediately above the inguinal ligament and is prolonged upwards and backwards above the iliac crest according to the point at which it is intended to divide the pelvic girdle; it is carried downwards across the pubic tubercle into the crease between the adductor region of the thigh and the perineum. The posterior part of the skin incision is made later in the operation, but is marked out at the beginning for convenience. It runs from the upper and posterior part of the first incision downwards, with a convexity forwards in front of the great trochanter, then winds round the posterior aspect of the upper thigh to meet the lower end of the anterior incision near the ischial tuberosity.

The antero-superior incision is deepened through the abdominal muscles above the iliac crest and in the groin, and the peritoneum is pushed medially from the iliac fossa and pelvic brim to demonstrate at the earliest moment the operability of the lesion. The spermatic cord is mobilised and displaced medially; the deep epigastric vessels are secured; the insertion of the ipsilateral rectus abdominis is divided, exposing the symphysis pubis; the bladder is separated from the back of this bone and protected by a pad, and the symphysis is divided with a knife—sometimes its complete dislocation at a later stage may require a little levering with a chisel. A ridge on the posterior aspect of the pubic bone in the median plane often provides a valuable guide to the exact site of the symphysis. The external iliac artery is identified and followed upwards, peritoneum and ureter being pushed medially until the common iliac vessels are exposed. The psoas is divided outwards from this point until the obturator nerve (under which the ilio-lumbar vessels lie) is seen; exposure of the common iliac vessels is thus facilitated. The common iliac artery is ligated and divided, the ilio-lumbar vein or veins are secured, and the common iliac vein mobilised, ligated and divided. The whole lower vascular pedicle is then pushed gently downwards until a finger can be introduced lateral to and above it into the apex of the sciatic notch.

The patient is now rolled gently into the full lateral position and the posterior skin flap is cut. Where the ilium is not involved the gluteus maximus is also in part reflected in the skin flap, and the gluteus medius is divided to expose the apex of the sciatic notch posteriorly. A pair of cholecystectomy forceps is thrust through the notch from behind forwards and upwards into the pelvis, emerging above and behind the distal segment of the divided vascular pedicle; by means of the forceps a Gigli saw is drawn through the notch and used to divide the ilium upwards and outwards to the junction of the middle and posterior thirds of the iliac crest. The pelvis can now be opened out, the patient being rolled dorsalwards; the pelvic viscera are pushed medially; the levator ani is divided; the crus penis and urogenital diaphragm are detached from the pubis. The laterally directed branches springing from the internal iliac vessels, comprising the external iliac, obturator, superior and inferior gluteal and internal pudendal vessels are secured; the sacrotuberous and sacrospinous ligaments are divided and the amputation is completed by severing the pyriformis and the sciatic nerve. The wound is closed by suturing the gluteus maximus to the anterior abdominal muscles.

Division of the symphysis pubis should be completed before any attempt is made to divide the posterior segment of the pelvic girdle. Even when this is carried out with a Gigli saw some laceration of the superior gluteal veins is inevitable, and though the iliac vessels have already been ligated fairly brisk haemorrhage may take place. If the symphysis has already been divided the whole pelvis can usually be opened out like an oyster shell as soon as the posterior bone section is completed, and haemorrhage is readily controlled by swabs pressed on the pelvic viscera. If the pelvis cannot be opened out because of postponed division of the symphysis, the haemorrhage following posterior bone section is difficult to control and much blood may be lost before the symphysis can be divided and separated.

If sacroiliac section with complete removal of the ilium is required, the posterior flap must be reflected further back to expose the posterior iliac spines and the sheath of the erector spinae; the flap consists of skin and subcutaneous tissue only, the gluteus maximus being left undisturbed. The erector spinae aponeurosis is divided immediately behind the posterior iliac spines and gives a clear indication of the required line of section. The incision

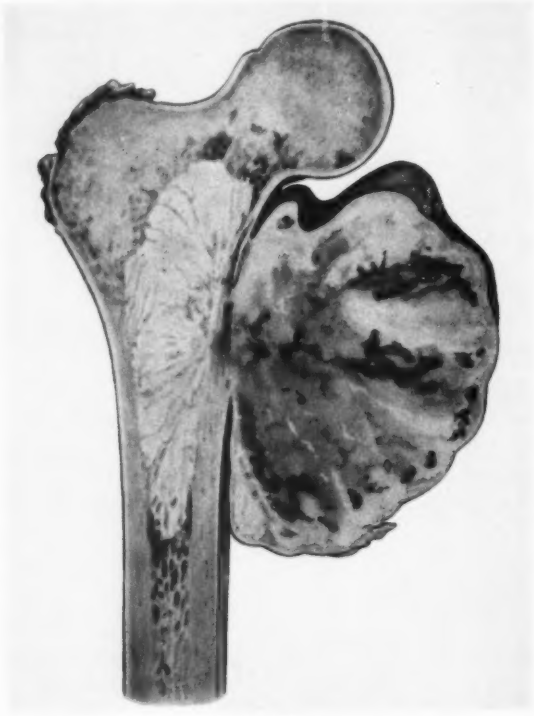

FIG. 3

Chondrosarcoma of the upper end of the right femur. The patient was a man aged sixty-four years, who made a good recovery from the amputation. A year after the operation prostatectomy was performed for adenoma.

is continued in its lower part into the origin of the gluteus maximus, and splits the muscle in the line of its fibres immediately below the posterior inferior iliac spine. The lower end of the Gigli saw is introduced in a manner similar to that already described, except that the forceps are thrust through the sciatic notch as close to the margin of the sacrum and sacrotuberous ligament as possible. Superiorly, the iliac crest is detached completely by excising the quadratus lumborum and erector spinae. The upper end of the Gigli saw can then be brought into the iliolumbar angle and across the anterior aspect of the sacroiliac joint to divide the iliolumbar ligaments and the pelvic girdle in the plane of the sacroiliac joint.

Occasionally the upward spread of a large neoplasm such as a chondrosarcoma of the ilium may have obliterated the space between the ribs and the iliac crest. Sacroiliac section is still achieved, but instead of the iliac crest and iliolumbar angle being cleared above as a preliminary to the saw cut, a modified Steinman pin is thrust from before backwards through the interspace between the fourth and fifth lumbar transverse processes. The upper end of the saw is attached

FIG. 4

Osteogenic sarcoma of the left hip bone. The patient was aged seventeen years, and made a good recovery from the operation. There was a recurrence on frontal bone one year after operation.

to the butt of the pin and is drawn through to the back, so that once more the saw lies across the front of the sacroiliac joint, with its two ends projecting posteriorly.

RESULTS

There have been eleven deaths in this series of fifty amputations, a mortality of 22 per cent; it is interesting to note that eleven amputations were carried out on patients over the age of fifty, with only one death. Death has been attributable in most cases to shock and haemorrhage. There has been a notable reduction in mortality in the last decade, though

only one quarter of the cases before 1940 required disarticulation and in the next decade it was necessary in one third (Table II). Twenty-two of our patients are alive to-day. Ten of them have survived five years, and many survived five years before dying of a recurrence of their disease (Table III).

TABLE II

MORTALITY

Category	Total cases	Deaths	Mortality per cent
All amputations	50	11	22
Amputations before 1940	11	4	36
Amputations after 1940	39	7	18
Amputation with sacroiliac disarticulation	17	5	29
Amputation with iliac section	34	6	18

TABLE III

RESULTS

Amputations	50
Patients now alive	22
Patients surviving more than five years	10

Results classified according to disease. *Chondrosarcoma*—Number of cases, 15; deaths due to operation, 1; patients still alive, 10; patients surviving more than five years, 5; one patient has survived twelve years. *Spindle-cell sarcoma*—Number of cases, 10; deaths due to operation, 1; patients still alive, 2; one patient has survived two years, and one has survived sixteen years. *Fibrosarcoma*—Number of cases, 9; deaths due to operation, 3; patients still alive, 2; one patient has survived five years. *Osteogenic sarcoma*—Number of cases, 5; deaths due to operation, 2; patients still alive, 3; one patient has survived seven years. *Osteogenic sarcoma complicating Paget's disease*—There have been two patients; one died immediately after operation, the other survived for one year. *Ewing's tumour*—There have been two cases; one died immediately after operation, the other is still alive eighteen months after operation, though a local recurrence necessitated radiotherapy. *Round-cell sarcoma*—There have been two patients; one died immediately after operation, the other is still alive nine months after operation. *Osteoclastoma*—There has been one patient, who is still alive twenty-two years after operation. *Angioendothelioma*—There has been one patient, who is still alive two years after operation. *Myosarcoma*—The one patient died nine months after operation. *Carcinoma of vulva with secondary deposits*—There has been one patient, who is alive five years after operation. *Osteomyelitis*—There has been one patient, who died two weeks after operation.

A minority of the patients have survived for many years after removal of what would otherwise have been a fatal lesion; the operation is thus more than justified despite its high mortality rate and inevitable mutilation. What the shorter survival figures fail to show is the relief from pain which the procedure usually affords as well. The contribution to the patients' happiness and well-being is considerable, even though death from pulmonary metastases may occur a few months later. It is to be regretted that in some cases reasonable

FIG. 5

Osteogenic sarcoma of the upper end of the right femur. Pathological fracture of the neck can be seen. The patient made a good recovery after amputation.

prospect of cure has been denied the patient because amputation has been undertaken only after repeated and inadequate local excisions. In others there has sometimes been no real attempt at cure at all and amputation has initially been passed over in favour of a palliative operation, or of radiotherapy in cases which were resistant to this treatment. Such surgical dilettantism reduces the chances of effective amputation.

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REPOSITION WITH ARTHROPLASTY FOR CONGENITAL DISLOCATION OF THE HIP IN ADULTS

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Open reduction of dislocation of the hip in the adult has been attempted by several workers. The poor results obtained by Lorenz (1892), Kirmisson (1898) and others were improved with the technical progress of arthroplasty, which was advised before the war by Galloway (1926), Samson (1938) and Tavernier (1919). Nevertheless, the procedure has remained an unusual one, because of its severity and the poor mobility it usually achieves. The interposition of inert material between the joint surfaces may improve the results. Smith-Petersen (1948) reported forty cases of arthroplasty, but only twelve have been reviewed in detail by Law (1948): in four the results were satisfactory, in five a second operation was necessary and in three the results were poor. Stinchfield and Carroll (1949) have reported five cases with three good results.

This paper is based on thirty-two operations in twenty-eight patients. All were over eighteen years of age; two were under twenty, fifteen were between twenty and thirty, six were between thirty and forty, and five were over forty.

Anatomy. *Side affected*—One in twelve patients; both in sixteen patients (operation on both sides in four). *Degree of dislocation*—Complete dislocation in all cases; in one case, one hip was dislocated and the opposite hip subluxated. *Position of the head relative to the acetabulum*—Anterior in six cases, high posterior in eighteen and intermediate in eight (Table I). *Function*—The operation was proposed only to those patients having severe interference with function. Patients able to walk one hour without severe pain were not accepted for operation. Assessment was sometimes difficult, because young girls who were anxious to get rid of their limp tended to exaggerate their pain and disability.

Pre-operative traction was thought useless, and omitted. All operations were performed under general anaesthesia, on an ordinary operating table without screw traction apparatus.

OPERATIVE TECHNIQUE

In eighteen operations the Smith-Petersen anterior approach was used. In ten, the ilio-psoas muscle had to be cut obliquely to allow exposure and deepening of the acetabulum; the femoral nerve was defined and protected. In twelve operations, the postero-lateral approach described by Gibson (1950) was employed, and it was found more convenient: it gives a better view of the capsule and acetabulum, the psoas is not a hindrance, and the trochanter may be lowered at the end of the operation. In two operations Ollier's (1885) incision was used. The whole capsule was excised. The cavity of the acetabulum, always small, was enlarged posteriorly and superiorly; atrophy of this part of the iliac bone frequently made it difficult to obtain an adequate cavity without risk of a fracture. The anterior and posterior margins of the acetabulum were clearly exposed so that it could be deepened at the correct point. In nearly half the cases the antero-posterior diameter was too small to provide a cavity able to receive a vitallium cup even of the smallest size. The use of an acrylic head allows of a smaller cavity. In two operations no material was interposed between the femoral head and the acetabulum. In one case the capsule was used, as described by Colonna (1947), with a good result. A Smith-Petersen cup was applied in sixteen cases, a Judet's acrylic head in thirteen (Table II). This material seems particularly useful when the head and neck of the femur are small and atrophic, because it restores some length to

this part of the femur. Whatever material is used, it is essential to resect as little of the femoral head as possible.

Reduction is sometimes surprisingly easy; in other cases strong and prolonged leverage is necessary. Only in two cases was it impossible to bring the head down, and the acetabulum

TABLE I

CLINICAL FEATURES IN TWENTY-EIGHT PATIENTS (THIRTY-TWO OPERATIONS)

AGE				
Number of patients	Under 20	20 to 30	30 to 40	over 40
28	2	15	6	5

SIDE			
Number of patients	Unilateral	Bilateral	
		Operation one side	Operation both sides
28	12	12	4

POSITION OF HEAD BEFORE OPERATION			
Number of operations	Anterior	High posterior	Intermediate
32	6	18	8

TABLE II

TECHNICAL DETAILS OF THIRTY-TWO OPERATIONS

INCISION				
Number of operations	Smith-Petersen		Gibson	Ollier
	Ilio-psoas cut	Ilio-psoas not cut		
32	10	8	12	2

INTERPOSITION					
Number of operations	None	Capsule	Cup	Acrylic	Diaphysial resection
32	2	1	16	13	4

had to be cut a little higher. Once a diaphysial resection was necessary. The stability of the joint must be carefully assessed because there is a danger of recurrence immediately after operation. A wedge-shaped shelf made with a piece of the femoral head or of the iliac bone and fixed with a screw is a useful means of completing the anterior part of the roof of the acetabulum. The greater trochanter is lowered by one or two centimetres and fixed with a wire.

Post-operative care—Immobilisation in plaster is necessary for three to four weeks. The limb must be placed in full abduction and neutral rotation. In my first cases the limb was immobilised in medial rotation for the sake of stability, but this led to permanent limitation of lateral rotation. In later cases, careful deepening of the acetabulum, combined with a screwed-in shelf and lowering of the trochanter, ensured enough stability for the plaster to

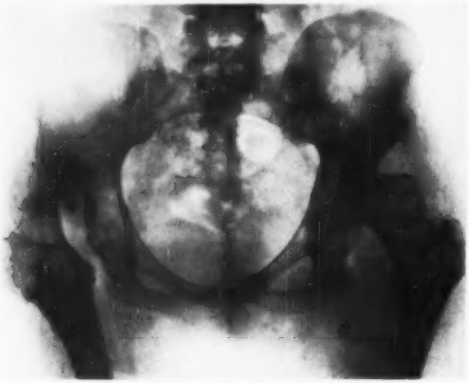

FIG. 1

FIG. 2

Case I—Aged twenty-five years. Figure 1—Congenital dislocation of right hip, with severe pain. Left hip subluxated and painful. Walking only a few yards. Figure 2—Arthroplasty, osteotomy and nailing of femur on the right side. Arthroplasty on the left hip six months afterwards. Eight months later there was good mobility and no pain in the right hip, and good mobility, but slight pain on the left.

be applied in neutral position. Two weeks after removal of the plaster walking with crutches was allowed, but full weight-bearing was delayed till the end of the fourth month. When a bilateral operation seemed necessary the patient was usually sent home as soon as he was able to walk with a stick, and full rehabilitation was awaited before the second side was treated.

RESULTS

Complications—No patient died. In three cases, shock made prolonged resuscitation necessary. *Redislocation* occurred in seven cases. Closed reposition was possible in three cases, but a second redislocation in two of these required open operation with deepening of the acetabulum and application of a shelf. In two other cases open reposition was performed

FIG. 3

FIG. 4

Case 2—Aged eighteen years. Figure 3—Bilateral congenital dislocation. There was little pain but walking was limited. Figure 4—After bilateral arthroplasty with an acrylic prosthesis and the construction of a shelf from the femoral head. There was good mobility and stability on the right side four months after the first operation; three months after the second the patient was beginning to walk.

without previous recourse to closed reduction. *Thrombophlebitis* occurred in two patients; one had an embolus, but this left no residual symptoms. *Sciatic pain* was observed in six cases after a high dislocated head had been brought down. The pain later disappeared, but sometimes only after three or four weeks. No femoral or sciatic paralysis was observed in

TABLE III

IMMEDIATE RESULTS

Thirty-two operations on twenty-eight patients

Mortality	nil
Anatomical result:	
Head in exactly normal position	12
Head slightly higher	14
Head higher than normal but stable	6
Complications:	
Redislocation: closed reduction	3
Redislocation: open reduction	4
Thrombophlebitis	2
Sciatic pain (tempo: 1 y)	6
Permanent medial rotation	4

TABLE IV

METHOD OF GRADING FUNCTIONAL VALUE OF THE HIP

	Painlessness	Mobility	Ability to walk
0	Pain intense and permanent	Ankylosis with bad position of the hip	Unable to walk
1	Pain severe even at night	No movement with pain or slight deformity	Only with crutches
2	Pain severe when walking preventing any activity	Flexion under 40°	Only with two sticks
3	Pain intolerable with limited activity	Maximum flexion between 40° and 60°	With one stick, less than one hour. Very difficult without stick
4	Pain mild when walking, disappearing with rest	Maximum flexion between 60° and 80°. Patient can reach his foot	A long time with a stick. A little without stick but with limp
5	Very mild and inconstant pain permitting normal activity	Maximum flexion between 80° and 90°. Abduction at least 15°	Without stick but with slight limp
6	No pain	Maximum flexion more than 90°. Abduction to 30°	Normal

TABLE V

FUNCTIONAL RESULTS

Fourteen operations before January 1950

Very good: 4 operations	No pain
	Walking without a stick with slight limp—2
	Walking with a stick (but could not walk before)—2
Good: 8 operations (6 patients)	No or negligible pain
	Flexion range 60 to 80 degrees
	Use a stick out of doors
Poor: 2 operations	Persistent pain
	Average flexion range 60 degrees
	Less able to walk than before operation

this series. *Permanent medial rotation* up to 10 degrees has impaired the result in four of my early patients. Four others have recovered the neutral position but cannot rotate the femur laterally. This trouble can be avoided by immobilisation in the neutral position after operation.

Anatomical results—Post-operative radiographs showed the femoral head placed exactly normally in twelve, slightly higher in fourteen, and elevated but apparently normal in six (Table III).

FIG. 5

FIG. 6

Case 3—Aged nineteen years. Figure 5—Congenital dislocation of left hip, previously treated by osteotomy. There was shortening of eight centimetres, pain and severe limp. Figure 6—Cup arthroplasty, osteotomy and nailing of the femur. Ten months later there was shortening of three centimetres, no pain, and slight limp.

FIG. 7

FIG. 8

Case 4—Aged twenty years. Figure 7—Congenital dislocation of the left hip. Figure 8—after cup arthroplasty. The patient had good mobility and was without a limp two and a half years after operation.

Functional results have been assessed by the method of grading that I use for all hip operations (Table IV). The three essential elements—painlessness, mobility, ability to walk—are graded from 0 to 6 (6 being normal, or nearly normal) both before and after operation. The benefit of the operation is assessed by taking the difference between the pre- and post-operative numbers, giving a double value to the first (pain) and third (walking) numbers, mobility being considered less important.

Progress after all hip arthroplasties is slow, but rehabilitation after arthroplasty for congenital dislocation is particularly prolonged. The walking ability of some patients is still improving after eighteen months or two years. The results can therefore be assessed of the first fourteen operations only, which were performed on twelve patients before January 1950. Of these, four represent *very good results*: two, operated on in November 1948 and January 1949, walk without a stick and with a slight limp, with good mobility and little pain; the two others were unable to walk before operation, and walk now with a stick and without pain. Six patients, on whom were performed eight operations, represent *good results*: pain is completely or nearly relieved, flexion varying from 60 degrees to 80 degrees is preserved, and they

TABLE VI

INFLUENCE OF OPERATION ON PAIN AND MOBILITY

	Before operation		After operation	
Pain (19 patients)	Painless	1 patient	Still painless	1
	Painful	18 patients	Worse	0
			Unchanged	2
			Diminished	10
			Completely relieved	7
Mobility (23 patients)	Mobility normal or more	16 patients	Greatly reduced	4
			Reduced	7
			Preserved	5
	Limited mobility	7 patients	Reduced	0
			Unchanged	2
			Increased	5

walk well with a stick. All of them are progressing. At the present time two represent *bad results*: pain is persistent, and, in spite of good movement, ability to walk is less than before operation. Cup arthroplasties were done in both, and the reason for the persistent pain seems to be penetration of the trochanter by the cup because of shortness of the neck. These patients could probably be improved by removal of the cup and substitution of an acrylic head.

Effect of the operation on symptoms. *Pain*—Only one patient had no pain before operation and she remained pain-free afterwards. Among nineteen others, pre-operative pain was unchanged in two, diminished in ten and relieved in seven. *Mobility* was assessed in twenty-three patients. Sixteen had a normal or exaggerated mobility of the affected hip or hips before operation; this mobility was greatly reduced in four (flexion limited to 60 degrees), slightly reduced in seven (flexion limited to 80 degrees), and unchanged in five. In seven patients with limited mobility before operation, the mobility was unchanged in two and increased in five (Table VI).

CONCLUSIONS

Arthroplasty, with normal or nearly normal reposition, is possible in most old congenital dislocations of the hip in young adults. Reposition is possible even in very high dislocation, but it is difficult or impossible in aged patients and when arthritic changes have taken place. The reduction equalises the length of the extremities in unilateral cases, but makes operation on both sides necessary in bilateral cases. Post-operative complications may be largely avoided by a good technique and careful post-operative treatment. Stability is good, and consequently the limp is generally reduced. Mobility is as a rule sufficient, though often less than before operation when the hip was normally or excessively mobile. Pain is very

often relieved or greatly reduced, but walking is generally limited. Even if the radiographic appearance of the hip is almost normal, the functional result may be imperfect.

FIG. 9

FIG. 10

Case 5—Aged thirty-two years. Figure 9—Congenital dislocation of the left hip. There was severe pain and little mobility. Walking was limited. Figure 10—After arthroplasty with an acrylic head and the construction of a wedge-shaped shelf from the femoral head. Six months after operation there was good mobility and no pain. The patient still required a stick.

This assessment is probably pessimistic, for two reasons: first, patients operated on are still improving; second, the improved technique of the later operations should give better results. Nevertheless, arthroplasty with reduction should be proposed to patients only suffering severe functional disability.

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SPRUNG BACK

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These observations were made in a backache clinic to which all patients had been referred by the staff of the hospital. For this reason backache due to infective and neoplastic disease had been eliminated and the symptoms in most patients were due to mechanical causes. The main object of the clinic so far has been to separate the different clinical syndromes and to try to find an explanation of their origin. One group, which last year amounted to about 20 per cent of all cases, has become distinct and I have termed it "sprung back." This paper describes the syndrome and attempts to explain its etiology and pathology.

THE CLINICAL PICTURE

The patient is usually between the ages of fifteen and thirty-five years and is more commonly a female. She complains of pain across the lumbo-sacral area, sometimes lower. The pain often radiates to one or both buttocks and to the back of the thighs and occasionally extends farther down the limbs. It is described as a dull ache, a nagging pain or a discomfort, but not often as a sharp pain. There may be exacerbations from time to time but the pain is seldom severe enough to necessitate prolonged rest in bed. The symptoms are not ordinarily dramatic but are productive of irritation and fatigue, impairing the joy of living. In some cases an attack of acute sciatica supervenes. The patient often attends many departments of the hospital for investigation and, being unimproved by treatment, may finally be referred to the psychiatrist who, casting his bait upon troubled waters, lands a "red herring."

The onset may be associated with a fall or a blow on the back, a strain while lifting something heavy, childbirth, or violent activity during adolescence; but sometimes there is no idea as to the cause. Many of these patients are young housewives, nurses or young men doing occasional heavy manual work to which they are unaccustomed. They complain that the ache is worse in certain positions, and in circumstances in which the lumbar spine is flexed and is either subjected to strain or is held in one position for a long time. Examples are: 1) Sitting in a slouching position with the lower part of the back unsupported. This causes aching pain which can be relieved by sitting straight with a cushion placed in the small of the back. 2) Lifting a weight at arm's length. 3) Bending and stooping, especially with straight knees. Standing with a slight forward stoop for a long time is followed by a feeling of stiffness when the upright position is resumed. Such a posture is often adopted by women when washing, ironing and cooking. Pain may also be felt when waking after sleeping curled up on the side, or when lying flat on the back with the legs straight.

Physical findings—In the standing position the lumbar curve is well marked and there may be some muscle spasm, but this not severe. Active flexion of the lumbar spine may be slightly limited, but generally, like other movements, it is full. Straight leg raising may cause pain by rotating the pelvis but not by stretching the nerve roots. There is no evidence of impairment of nerve-root conduction. With the patient prone and three or four pillows beneath the abdomen she can relax into a semi-flexed position (Fig. 1). This opens up the gap between the spinous processes and facilitates palpation of the supraspinous ligaments. Tenderness is found in the mid-line either at the space between the spinous processes of the fifth lumbar and first sacral vertebrae, or between the fourth and fifth lumbar vertebrae. Frequently a depression, denoting damage to or laxity of the supraspinous ligament, is felt

FIG. 1
Method of examining the supraspinous ligament.

at one of these interspaces and contrasts markedly with the resistance of the intact ligament at other levels. Small firm nodules can sometimes be palpated at the site of tenderness and are a guide to the level of the lesion.

A useful diagnostic test is to inject fluid into the suspected interspinous ligament after the manner of Kellgren (1939), when the pain of which the patient complains is intensified. If a local anaesthetic is injected there is first an initial increase of pain due to tension and then it is temporarily relieved.

Radiographic examination—The usual antero-posterior, lateral and oblique views give little help, but lateral views of the spine in full extension and full flexion may show mechanical instability.

ANATOMY

The spinous processes of the lumbar vertebrae are well adapted to act as levers which enable the muscles and ligaments attached to them to control easily the "set" of the vertebral bodies in front. They are strong and well separated from their fellows as they protrude posteriorly. Each is enlarged at its tip; the enlargement is greater below, where there is a bony prominence at the inferior angle on each side, giving a rather pear-shaped appearance when seen end-on from behind (Fig. 2). These prominences are for the attachment of the tendons of the multifidus muscles which are responsible for extending each vertebra individually and which also supply the braking mechanism when the spine is flexed in the standing position. The erector spinae mass extends the vertebral column as a whole, but the column consists of individual units dependent on each other so that, like members of a rowing crew, each must pull its weight. A vertebra without a spinous process, like an oarsman without an oar, is carried along by his fellows but not without detriment to the general efficiency of the crew.

The lumbar fascia of each side meets in a median raphe which is attached to the posterior and lateral surfaces of the tips of the spinous processes and is known as the supraspinous ligament. This is well developed and strong, being at least as thick as the top of the individual's fifth finger. The supraspinous ligament acts as the main check to overflexion of individual pairs of vertebrae and of the whole spine. It is placed, like the tendons of the multifidus, at the extreme end of the lever where its power is most effective.

The interspinous ligaments and joint capsules are less strong than the supraspinous ligament. The ligamentum flavum, although thick, contains much elastic tissue and stretches

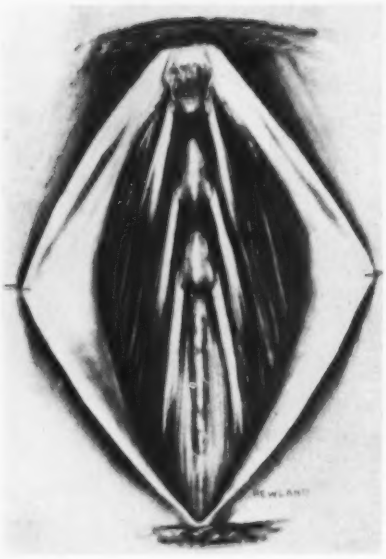

FIG. 2

Drawing of a dissection in which the lumbar fascia has been reflected and the supraspinous ligament removed. It shows the enlargement of the spinous process inferiorly where the tendons of the multifidus are attached.

rather than tears under stress. Its elasticity is said to give posture to the column and to protect the dura. The elasticity also prevents infolding during full extension, with its possible harmful pressure on the dura.

FLEXION INJURIES OF THE VERTEBRAL COLUMN

The ramifications of idea and thought on the subject of backache are so extensive, and so many are the possibilities of investigation, that discipline is essential lest the issue be confused and lost in a mass of experiment and surmise. Since one object of this paper is to show that overflexion of the lower lumbar spine primarily injures the posterior structures of the vertebral column at the same level, the argument will be directed only to that end.

Forced flexion of the vertebral column as a whole may cause injury to the anterior structures—that is, to the vertebral bodies—or to the posterior structures, such as the fascia, ligaments and joint capsules and sometimes also the neural arch. Anterior and posterior structures at either the same or different levels may be damaged simultaneously. Which of these events occurs is determined by the extent and direction of the violence, the anatomical arrangement of the particular part of the spine concerned and the posture at the moment of impact.

Injury to anterior structures—Jefferson (1927) reported 2,006 cases of fracture of the vertebral column and a graph has been constructed from his figures showing the incidence of injury to each vertebra (Fig. 3). Most fractures occurred in the low cervical region (C.5, 6, 7) or in the thoraco-lumbar region (T.11, 12; L.1, 2). The upper ten thoracic vertebrae are damaged relatively infrequently. The vertebral column here is less mobile, the spinous processes are long and lie over one another bound together by strong ligaments, and the true ribs are attached to the transverse processes behind and to the sternum in front. Where the ribs float and are not articulated anteriorly, movement of the vertebrae is freer.

In the neck the spine flexes so freely that the chin can touch the sternum, and this gives the cervical spine some protection from violent and sudden movement. When a flexion strain is applied that is too powerful or too sudden the spine breaks; and it breaks where one would expect, at the level where the mobile cervical vertebrae meet the immobile thoracic vertebrae. The sixth cervical vertebra is the most vulnerable because the seventh with its long spinous and

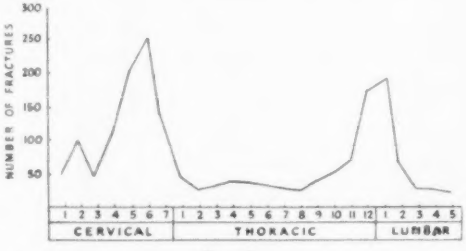

FIG. 3

Incidence of vertebral fractures at different levels of the spinal column in 2,006 cases (Jefferson).

transverse processes is relatively immobile. The nature of the injury depends on the integrity of the ligamentum nuchae; if it remains intact there is a crush fracture, but if it ruptures a dislocation usually occurs as well.

Excessive flexion violence applied to the trunk fractures the vertebral column just below the thoracic cage, seldom in the lower lumbar region. The reports of Jefferson (1927) and of Nicoll (1949) (Fig. 4) show that the highest incidence of fracture is at T.12 and L.1. The incidence at each vertebra below this level falls rapidly, and in the lower three lumbar vertebrae it is little more than one-tenth of that at L.1. It is not at first obvious why the lumbar spine breaks at its upper part. On the analogy of the cervical spine it might be expected to yield where the last mobile vertebra meets the pelvic girdle. The cervical spine, however, has a range of flexion that allows its curve to be reversed, and each vertebra down to the sixth or seventh joins in the movement. On the other hand, flexion of the lumbar spine is restricted by the posterior ligaments and by the "set" of the joint facets; moreover, when the knees are flexed to relax the hamstrings, the pelvis itself is able to move. Reversal of the lumbar curve to one of flexion can be demonstrated radiologically only in acrobatic dancers and in other very supple people. A flexion force applied to the trunk causes a crush fracture at the site where greatest flexion occurs and this is at the upper lumbar region. The lower lumbar vertebrae are protected by the lordotic curve, and before one vertebral body can impinge on and crush another the posterior structures must give way. It is, in fact, quite common for the posterior ligaments of the lower lumbar region to be torn by the same injury that produces a crush fracture of an upper lumbar vertebra. This accounts for low lumbar pain persisting long after a fracture at a higher level has become symptomless.

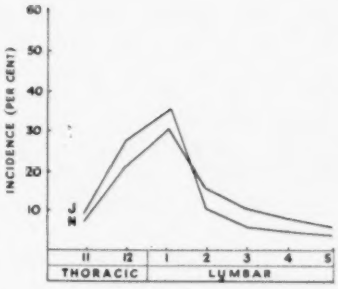

FIG. 4

Incidence of vertebral fractures at different levels of the thoraco-lumbar spine in two series of cases. J = Jefferson (1927), N = Nicoll (1949).

Injury to posterior structures—Nicoll (1949) pointed out that if the knees are extended at the time of injury the pelvis is held rotated backwards by the tight hamstrings and greater stress falls on the lumbo-sacral region. He mentioned two cases of traumatic spondylolisthesis of the fifth lumbar vertebra occurring in this way. Griffiths (1949) suggested that crush fracture may occur at the lower lumbar level when the patient has warning of the approaching blow and has time to brace himself against it, as when a man is standing back to a stack of bales, or to a side wall of a trench and senses its imminent collapse. It is understandable that the vertebral column in these circumstances breaks at a lower level because the pelvis is fixed by the tight hamstrings and the lumbar curve is forced into flexion after rupture of the soft structures posteriorly (Fig. 5). This type of accident is uncommon, but less severe strains to the lumbar spine, with the knees in full extension and the hamstrings taut, are common. The violence may be sufficient to rupture some or all of the posterior ligaments, including sometimes the posterior longitudinal ligament and the annulus fibrosus. The back has then a permanently weak

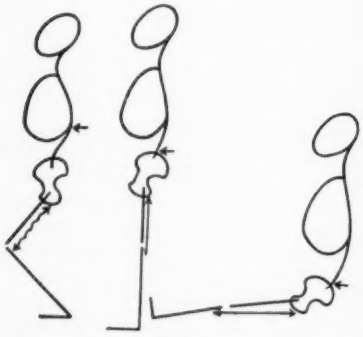

FIG. 5

Three ways in which the spine is injured by flexing force. When the hamstrings are relaxed the pelvis is not fixed and injury will usually occur at the thoraco-lumbar junction. When the pelvis is fixed injury is likely to affect the lumbo-sacral area.

and painful segment which has resulted from the springing apart posteriorly of two vertebrae. It can be described simply as "sprung back."

Injuries of this sort occur frequently in everyday life, as in a fall on a slippery surface with the legs stretched out in front, a fall downstairs, a fall from a horse or bicycle or when skating, a fall when a chair is jokingly pulled away from a person who is about to sit down. Usually the arms are held out behind to break the force, but if the hands are otherwise occupied, or are slow in their reaction, the full force of the blow is taken by the pelvis and lumbar spine at a time when the protective lordotic curve of the spine is obliterated. A similar strain to

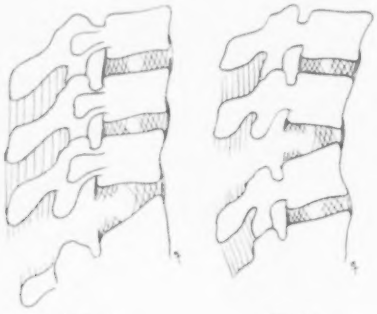

FIG. 6 FIG. 7

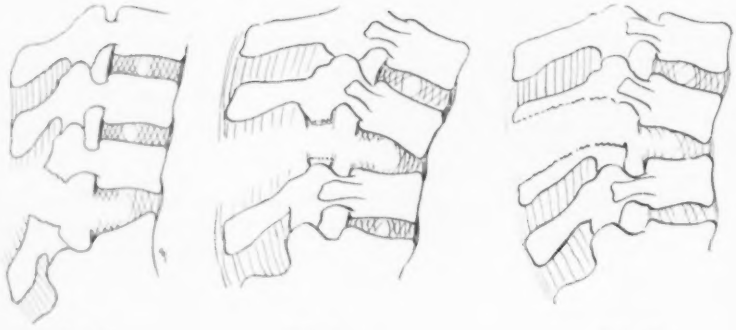

FIG. 8 FIG. 9 FIG. 10

FIGS. 6 to 10

Five types of injury to the posterior structures of the lower lumbar spine. Figure 6—Springing of L.5–S.1 space. Figure 7—Springing of L.4–5 space. Figure 8—Fracture through the pars interarticularis. Figure 9—Fracture through the facet and springing of the L.4–5 space. Figure 10—Longitudinal fracture through the spinous process.

the spine occurs when a heavy weight is lifted with a flexed spine but straight knees; it occurs also when the lumbar spine is flexed under an anaesthetic during a manipulation or during an operation in the lithotomy position, and even during a forceps delivery with the mother lying on her back. Operations, such as laminectomy, in which the spinous processes or their ligaments are removed, have the same effect.

The result of violence of this nature, when of sufficient force, is to tear the supraspinous and interspinous ligaments, the capsules of the joints and sometimes even the posterior longitudinal ligament and the posterior part of the annulus fibrosus. The ligamentum flavum, being elastic, stretches but does not rupture. The region of the spine affected is where the

mobile lumbar spine meets the immobile pelvic girdle, and this is generally the lumbo-sacral junction (Fig. 6). Sometimes the fifth lumbar vertebra is partly or completely sacralised or the transverse processes are unduly large, making it comparatively stable, in which case the rupture of the ligaments occurs between the fourth and fifth lumbar vertebrae (Fig. 7). On the other hand, if the transverse processes of the fifth lumbar vertebra are small and the spine of the first sacral element is ill-developed, or if there is a spina bifida, the posterior structures at this level are weak and are more likely to be damaged. Occasionally the capsules of the joints hold against the strain and the break traverses the pars interarticularis, causing a condition of spondylolysis (Fig. 8). More rarely there is a fracture of an articular facet (Fig. 9) and exceptionally the force may split a spinous process along its vertical axis (Fig. 10).

At the time of injury the back has "opened," and as a protective reaction the erector spinae muscles go into spasm. The torn structures often heal by fibrous union and the patient regains a comparatively strong back which gives rise to little trouble during the ordinary activities of life. On the other hand, the supraspinous and interspinous ligaments may fail to heal properly and remain in an elongated or even an interrupted condition, and the joint capsules may be lax. The affected vertebrae are then unstable and the joints may subluxate when the vertebral column is flexed or extended. In many cases it seems that the deficiency caused by the injury is filled by scar tissue, which gives rise to pain when it comes under strain. This occurs in full flexion, as when slouching in an easy chair, and also when a position of partial flexion is held for long enough for the erector spinae to become fatigued, as when standing over a sink.

Disc prolapse—It is part of the thesis that a flexion injury to the posterior structures of the vertebral column, if carried beyond the point at which the neural arches of two vertebrae are torn asunder, will separate the vertebral bodies and damage the posterior longitudinal ligament and the annulus fibrosus which bind the bodies together. It is rational to regard disc prolapse as secondary to ligamentous damage. Barr's (1951) comparison of a ruptured disc with a motor car tyre burst is indeed apt. Just as a tyre may burst at any time because of an inherent weakness, or burst at the actual moment of injury or not until thousands of miles after the injury, so may an intervertebral disc. But wherein lies the weakness of the back that causes chronic pain perhaps for years before prolapse of a disc takes place? Does it lie in the annulus fibrosus alone, as some believe, or does it involve all the structures which bind the vertebrae together posteriorly? From which structure does the chronic pain originate? There are certain pointers which lead one to believe that the injury to the annulus fibrosus is caused by posterior separation of two vertebrae, that the chronic weakness lies in all the structures binding the vertebrae together posteriorly, and that at least a great deal of the pain arises from the supraspinous and interspinous ligaments. This is suggested by the following evidence. 1) Injury to the posterior part of the disc occurs most commonly at the same sites at which flexion injuries damage principally the posterior structures (Fig. 11). 2) Friberg and Hirsch (1949) have shown by the examination of 500 discs at necropsy that in the lower lumbar spine it is almost invariably the posterior arc of the disc which is damaged, and this suggests that the injuring force separates the vertebrae posteriorly. If injury to the

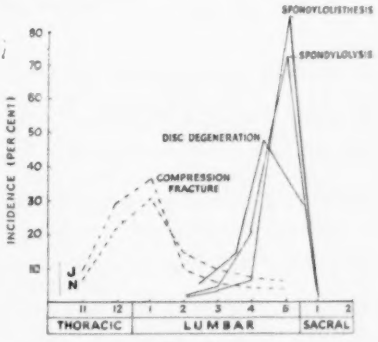

FIG. 11

The relative incidence of spondylolisthesis, spondylolysis, disc degeneration and fractures of the thoraco-lumbar region at different levels of the spine. J = Jefferson (1927), N = Nicoll (1949).

annulus were due to compression, torsion or grinding, the damage would not be confined to the posterior quadrant. 3) In my experience of operations for prolapsed disc, a torn or inefficient supraspinous ligament, and an unstable vertebra, are almost constant findings. 4) In cases of disc prolapse the supraspinous ligament is tender and deficient on palpation. 5) Chronic backache and attacks of acute lumbago frequently precede sciatica. 6) Kellgren (1939) showed that pain of a constant distribution could be produced by injecting hypertonic saline into the supraspinous and interspinous ligaments. When the ligament between L.4–5 spinous processes was injected, the pain was distributed over the buttock to the outer and anterior part of the thigh; and when the ligament between L.5–S.1 vertebrae was injected it was felt from the buttock, outer side of the thigh and calf to the foot. Pain of this distribution can usually be relieved by injecting procaine into the corresponding ligament.

Assuming that separation of the vertebrae has been sufficient to injure the annulus fibrosus, it is easy to understand how chronic backache and recurrent lumbago over a period of years can be converted suddenly to an attack of acute sciatica, with change of the clinical

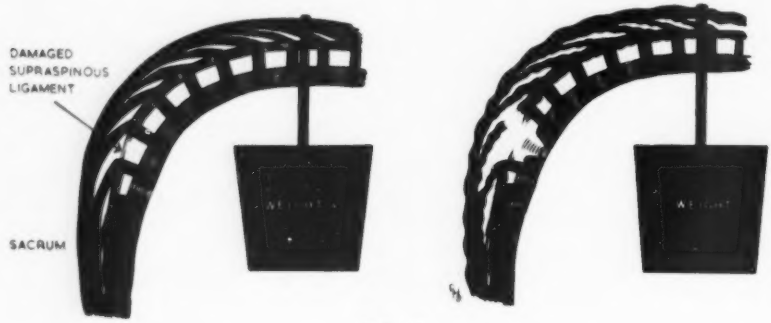

FIG. 12

To show the significance of a sprung lumbar space when the erector spinae muscle is inhibited during a time of considerable strain.

picture from one of moderate annoyance to that of an acute and incapacitating illness. The physical signs are altered by impingement of the prolapsed material on a nerve root. The straight-leg raising test becomes positive and the muscles of the back go into spasm. The posture, however, is one of flexion rather than extension, for extension would increase the extrusion. Lumbar spasm in flexion is as characteristic of disc protrusion as is spasm in extension of low back instability. The pain, formerly of scleratome distribution and relievable by an injection of procaine, now has a dermatome distribution and cannot be so relieved.

THE EFFECT OF SPRUNG BACK

A patient with a sprung back no longer has the ligamentous control necessary to prevent excessive flexion of the spinal column. In the normal spine the degree of flexion at each segment is "rationed" by the ligaments. In sprung back there is a break-down in the rationing process, the result being a painful, too liberal or too restricted range of movement.

Floyd and Silver (1951) have shown by electro-myographic studies that in the standing position there is no contraction of the erector spinae muscles, but as the spine begins to bend forwards these muscles contract until full flexion is reached. At this "critical point" the muscles again relax, leaving the spine supported by the ligaments. It is at this stage, when the spine is in a position of flexion with the erector spinae relaxed, that the ligaments of a damaged segment break down at their job and either fail to take the strain or take it painfully. This may necessitate continuous contraction of the erector spinae muscles, which consequently

become fatigued. If a flexed position is maintained for some time there is aching and stiffness when the upright position is resumed. Sometimes there is a sharp pain during recovery, even in the absence of undue strain, but if force is applied when the spine is flexed and the erector spinae relaxed, an acute attack of pain is more likely to occur (Fig. 12). Acute pain is also liable to occur when a heavy weight is lifted, or when an object is lifted hurriedly. The gardener who anticipates lifting a barrow-load of earth does not rush at it; he stands between the handles, prepares his muscles for action, bends his knees and gently lifts the barrow with his quadriceps and glutei. A sudden movement such as taking a low volley on the tennis court, saving a child from falling into the fire, or attempting to recover after slipping while carrying something heavy in the arms, is more likely to cause an acute attack than is a deliberate, controlled movement. The erector spinae muscles can also relax, at least over part of their length, when the spine is held in mid-flexion, for I have knowledge of three patients who have had an acute attack of lumbago while shaving. In this position the spine relaxes while flexed forward over a wash-basin and the head is poked out to view its image in the mirror, and on sudden recovery there may be an acute attack of pain. Thus a patient with sprung back is subject to chronic backache and to acute attacks of lumbago; and he may also become the victim of acute sciatica. The causes of chronic backache must be almost as numerous as those of abdominal pain, but in my opinion the commonest of all is strain on a segment of the spine after the normal checking function of the ligaments and capsules has been impaired by injury. Should a new force stretch the ligaments still further, the guardian muscles at once go into action giving rise to spasm in extension. This is the rational response of a segment that has been damaged by being forced beyond its limit of flexion. It can be described simply as lumbago in extension.

SUMMARY

Sprung back is one of the commonest causes of low back pain. Its characteristic features are described. It is caused by rupture of the posterior ligaments of the spine, including sometimes the posterior longitudinal ligament and annulus fibrosus. The manner in which it is produced and its mechanical effects are discussed in detail.

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LOCALISED OSTEOCHONDRITIS (SCHEUERMANN'S DISEASE)

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The multiple form of Scheuermann's disease (adolescent kyphosis; osteochondritis vertebralis juvenilis) affecting the thoracic spine is well recognised, but there is a localised form occurring in the thoraco-lumbar region which, although no doubt well known, does not seem to have attracted so much attention. The only account of it that I have found is that of Wiles (1949) who refers to it as "dorso-lumbar kyphosis." It is important that this condition be distinguished from tuberculosis, with which it is easily confused in the early stages. The clinical similarity may indeed be close, but once the radiographic appearances are recognised the difficulty in distinguishing between the two conditions largely disappears.

This paper records four cases of localised osteochondritis, all in adolescents, their ages varying from fourteen to seventeen years at the time of onset of symptoms. All complained of pain in the thoracic-lumbar spine, and in one case the pain was referred to the loin. In three of the four patients a slight kyphosis could be seen and felt in the thoraco-lumbar region. There was tenderness on palpation over the affected vertebrae. Muscle spasm was slight or absent and there was no muscle wasting. Movements of the spine were limited, especially forward flexion. The symptoms subsided rapidly with immobilisation of the spine in bed or in a plaster jacket or brace.

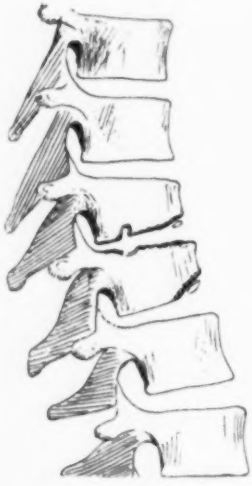

FIG. 1

Diagrammatic representation of the radiographic appearances in localised osteochondritis. The characteristic features are wedging of two adjacent bodies, Schmorl's nodes, diminution of the intervertebral disc space, fragmentation of the epiphysial rings, and sclerosis of the adjacent vertebral margins.

Radiographic examination—The radiographic appearances are similar to those seen in adolescent kyphosis but confined to one region of the spine (Fig. 1). There is wedging of two adjacent vertebral bodies, often slight at first but well marked later. In patients seen before the secondary centres of ossification in the epiphysial rings have appeared the wedging is the only abnormality of the vertebral body. After ossification of the rings has begun the other features of osteochondritis, such as irregularity of the upper and lower epiphysial rings, Schmorl's nodes and sclerosis of the adjacent vertebral surfaces, appear. In all cases there is narrowing of the intervertebral disc space between the affected vertebrae, and it is this feature especially which suggests a diagnosis of tuberculosis. Bone destruction or rarefaction, either localised or generalised, is never seen, nor is there any evidence of abscess formation.

ETIOLOGY

The fact that the radiographic changes are like those seen in adolescent kyphosis suggests that the two conditions have a similar etiology. It may not be out of place, therefore, to review briefly the existing views concerning the bony deformity which occurs. The view most widely accepted is that there is primarily a lesion of the intervertebral discs resulting in herniation of disc material into the vertebral

bodies (Schmorl's nodes). This loss of substance results in increased pressure between adjacent vertebrae, the greater effect being anteriorly because the vertebrae are hinged behind at the intervertebral joint. When the secondary centres of ossification for the upper and lower epiphysial rings appear at about the time of puberty, they are subjected to pressure anteriorly and their growth is interfered with. This retardation of growth from pressure results in the wedging which is characteristically limited to the anterior part of the vertebral body.

An alternative view was suggested by Ferguson (1949), who considered that the condition can be traced to early infancy and is caused by exaggeration or persistence of the vascular pit and groove on the anterior and lateral surfaces of the vertebral body which normally are obliterated by the age of five or six years. Ferguson stated that in adolescent kyphosis the vascular groove persists into puberty or adolescence and is responsible for the wedging, the epiphysial changes being a secondary manifestation.

CASE REPORTS

Case 1—Boy aged sixteen years. Three months before he was first seen the patient experienced a sudden severe pain in the thoraco-lumbar spine after lifting a heavy weight. The pain had subsided with rest but had recurred when he returned to work. *On examination* there was a slight kyphosis in the thoraco-lumbar region. This area of the spine was slightly tender on palpation and there was limitation of flexion of the spine but other movements were full. There was no pyrexia, blood sedimentation tests were normal, and the Mantoux test was negative. *Radiographs* showed localised osteochondritis affecting the twelfth thoracic and first lumbar vertebrae (Fig. 2), but this was at first erroneously considered to be tuberculosis and the patient was treated by immobilisation on a plaster bed for three months. Serial radiographs showed no change. In view of the rapid disappearance of symptoms, the persistently negative tests and the absence of deterioration in the radiographic appearances, the diagnosis of tuberculosis was corrected and the case was regarded as one of localised osteochondritis. Progress was uneventful, and when last seen one year after the onset he was free from symptoms and at work doing a heavy job.

FIG. 2

Case 1—Localised osteochondritis affecting the twelfth thoracic and first lumbar vertebrae.

Case 2—Boy aged seventeen years. The patient was at work carrying a large piece of wood when he stumbled and felt a "crack" in his back. This was followed by considerable pain. *On examination* all spinal movements were painful and there was tenderness over the lower thoracic vertebrae. *Radiographs* showed localised osteochondritis of T.11 and 12. The symptoms settled down rapidly with rest. He was free from symptoms and was allowed to return to work six weeks later, but suffered a severe recurrence of pain and was readmitted. He was fitted with a Robert Jones back brace. When last seen one year later he was doing heavy work and had no symptoms. There was a full range of painless movement of the spine.

Comment—In view of the possible association between this condition and "congenitally short hamstrings," it is interesting to note that this patient is now able to touch his toes, a feat he had been unable to accomplish as long as he could remember.

Case 3—Girl aged fourteen years. When first seen by a general surgeon the patient complained of intermittent pain in the right loin over a period of three months. *On examination* there was tenderness in the right loin and an intravenous pyelogram showed no excretion on the right side. Cystoscopy was normal, but a retrograde pyelogram showed absence of the renal pelvis on the right side. Exploratory operation was undertaken and disclosed congenital absence of the right kidney. *Radiographs* of the thoraco-lumbar spine showed localised osteochondritis affecting

T.10 and 11 (Fig. 3). *Treatment*—The patient was fitted with a plaster-of-Paris jacket with relief of symptoms. When last seen three months later she was still getting some pain but had a full range of movement of the spine.

FIG. 3

Case 3—Localised osteochondritis affecting the tenth and eleventh thoracic vertebrae.

FIG. 4

Case 4—Localised osteochondritis affecting the first and second lumbar vertebrae.

Case 4—Boy aged fourteen years. The patient, a schoolboy, complained of pain in the lower thoracic spine, made worse by violent exercise, especially flexion of the spine. No kyphosis was present but there was a loss of the normal lumbar lordosis. Forward flexion of the spine was limited to about half the normal range, but other movements were free. Radiographs showed localised osteochondritis affecting L.1 and 2 vertebrae (Fig. 4). A plaster-of-Paris jacket was applied with relief of symptoms after one month. The jacket was retained for two months and the patient was advised to avoid games for a further month. When last seen eighteen months from the onset of symptoms he was still free from pain.

DISCUSSION

The facts that the symptoms are intermittent, that the radiographic changes are often observed in the absence of symptoms, and that the onset is associated with vigorous exercise or heavy work, suggest the possibility that the symptoms may not be due to the bony changes shown in the radiographs and that they may in fact be due to a posterior protrusion of the intervertebral disc. This was particularly suggested by Case 3, in which there was root pain. The radiographic changes may be merely coincidental or they may be consequent upon the intervertebral disc lesion in the manner which has already been discussed. From the point of view of treatment this matters little; the important thing to realise is that these changes are not due to an infective lesion.

I wish to thank Mr B. H. Burns and Mr R. H. Young for their permission to publish these cases treated under their care at St Peter's Hospital, Chertsey, and also for their encouragement and assistance. I am indebted to Miss Barbara Blakeway for the diagram.

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TRAUMATIC PREPATELLAR NEURALGIA

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Persistent investigation of a series of painful knees in which all the accepted methods of investigation were negative revealed a common point of tenderness towards the middle of the outer border of the patella. Careful dissection of the prepatellar [subcutaneous prepatellar] bursa in twenty-one such cases led to the definition of a syndrome characterised by the following features: 1) a history of well-remembered violence to the front of the patella, usually in a fall; 2) after the injury, sometimes transient prepatellar swelling, not circumscribed by

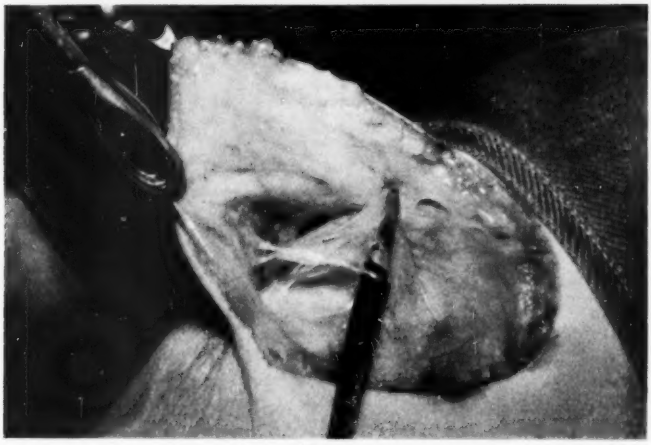

FIG. 1

The neurovascular bundle running from the deep surface of the skin to the bursal wall (supported by forceps).

the bursa; 3) immediately or several weeks after the accident the onset of neuralgic pain deep to the patella, described usually as a dull ache " like toothache "; 4) a precise point of tenderness (of which the patient may have been unaware) towards the middle of the outer border of the patella and nowhere else; 5) persistence of neuralgic pain and disability for many years—up to seven years in this series—with inability to kneel, difficulty in climbing stairs, difficulty in bending the knee and inability to walk far because of increasing ache in the knee; 6) frequent diagnostic difficulty from failure to associate the tenderness with the pain; 7) complete relief on removal of the neurovascular bundle which supplies the prepatellar bursa.

Thomas Walmsley (1934 *b*) gives the best description of the prepatellar bursa. He states: "The subcutaneous tissue over the front of the knee is thin, loose in texture, and nearly devoid of fat. Over the lower part of the patella and the upper part of the patellar ligament it contains a bursa, the largest subcutaneous bursa in the body. Its interior is intersected by fibrous bands which may subdivide it into three parts, namely: 1) a part between the skin and the subcutaneous tissue, 2) a part in the subcutaneous tissue, and 3) a

part in the lax connective tissue below the thick aponeurotic expansion of the quadriceps femoris which covers the front of the patella and is continued into the patellar ligament." Only the first and second parts are always present.

FIG. 2

Section of the neurovascular bundle, with considerable perineural fibrosis.

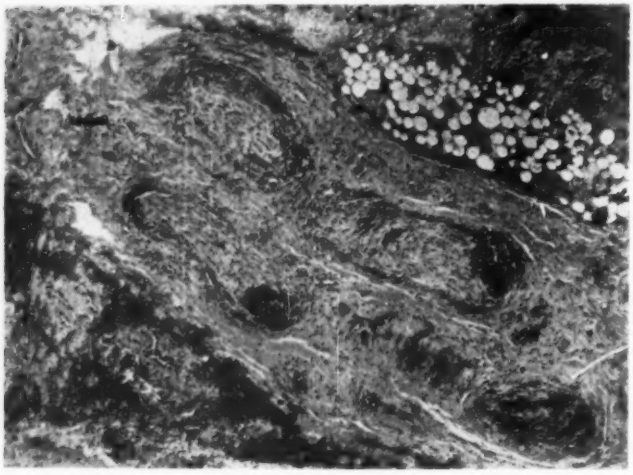

FIG. 3

Section of the neurovascular bundle in the case of strangulated tag, with haemorrhage into the tissue spaces and into the nerves themselves, and with gross perineural fibrosis.

Dissection in the present cases, frequently after a needle had been inserted into the tender point before anaesthesia, showed that there is a constant neurovascular bundle supplying the prepatellar bursa and issuing from the deep surface of the skin about the middle

of the lateral border of the patella (Fig. 1). There may be a single large contribution which branches over the bursal wall or many small contributions which are much more difficult to identify. After moderate intermittent pressure the subcutaneous part of the bursa may become so closely attached to the deep surface of the skin that it cannot be dissected free. It is in this area or close to it that the neurovascular bundle is found. Its removal, with a small part of the bursal wall, invariably relieved the original pain. Histological examination of the excised tissue, carried out in fifteen instances, revealed vessels and nerves (Fig. 2). The presence of a neurovascular bundle in this area was verified macroscopically and microscopically in two cadavers.

TABLE I

SUMMARY OF CASES

Case number	Age (*years*)	Occupation	Interval between injury and operation	Date of operation	Histological examination	Date of review	Residual symptoms
1	34	Housewife	4 months	31/7/50	Yes	10/2/51	None
2	28	Kitchen maid	2 weeks	10/1/51	Yes	17/3/51	None
3	51	Cleaner	9 weeks	13/2/50	No	13/2/51	None
4	29	Miner	5 months	20/3/50	No	10/2/51	None
5	31	Miner	4 months	15/6/50	No	10/2/51	None
6	46	Housewife	7 years	22/6/50	Yes	10/2/51	None
7	26	Paper mill worker	9 months	16/6/50	No	10/2/51	None
8	18	Warehouse assistant	6 years	31/7/50	Yes	10/2/51	None
9	24	Machinist	3 months	17/7/50	No	23/2/51	None*
10	55	Miner	8 days	7/9/50	No	7/2/51	None
11	49	Miner	5 weeks	7/9/50	Yes	7/2/51	None
12	44	Bakery assistant	1 month	16/9/50	Yes	21/3/51	None†
13	40	Housewife	5 weeks	11/9/50	Yes	10/2/51	None*
14	43	Housewife	4 years	25/10/50	Yes	23/2/51	None
15	23	Fitter	5 weeks	1/11/50	Yes	17/3/51	None
16	47	Housewife	10 months	15/11/50	Yes	23/2/51	None
17	15	Packer	5 weeks	31/11/50	Yes	17/3/51	None
18	27	Housewife	2¼ years	15/11/50	Yes	17/3/51	None
19	43	Housewife	2½ years	31/1/50	Yes	6/3/51	None*

* After injection of local analgesic into hyperaesthetic point in scar.
† After excision of hyperaesthetic point in scar.

The bursa may appear normal, or may show one of the following changes: obliteration by fine adhesions, caused by reaction to the injury; some thickening, with alteration of the fine trabeculae into dense fibrous cords traversing the bursa; or aseptic inflammatory changes, no evidence of which can be detected on the surface and which may have been present for some months. No fluid was found in any of the bursae dissected.

Seemingly part or all of the neurovascular supply to the bursa becomes involved in the reactive and healing processes after acute trauma, with consequent perineural fibrosis (Figs. 2 and 3) and the typical neuralgia.

Details of the nineteen cases of this series are shown in Table I. One case, in which a strangulated tag was found (Fig. 4), and one case of loose bodies, have been excluded from the series. The strangulated tag is a variant of the condition. Prolonged periods of kneeling lead to coarsening of the trabeculae, resulting in strands which may be avascular or contain part of the neurovascular bundle. When trauma involves the vascular type, strangulation occurs, as seen in Figure 4.

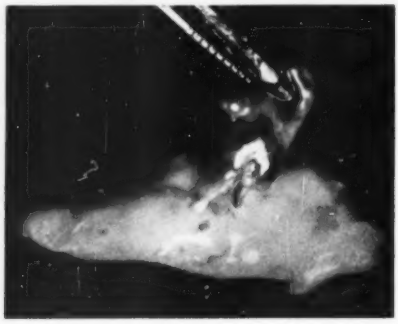

FIG. 4

Photograph showing an intensely congested strangulated tag. Note the fenestration in the bursal wall, from which the neurovascular bundle issues. The small horn seen near the neurovascular bundle was attached in the same way, but broke during manipulation.

Smillie (1946) has described a condition of "neuritis of a branch of the patellar plexus . . . assumed to be due to a minor direct injury which produces an irritative lesion of a minute sensory nerve." Apart from the pathology my cases differ from Smillie's in the characteristically constant position of the point of greatest tenderness, the onset of referred neuralgic pain, and the failure to recover spontaneously, in contrast to Smillie's experience that "although the condition may remain for a few weeks, it is self-limiting and will disappear as suddenly as it came."

I wish to acknowledge the constructive criticism and assistance given by Mr F. W. Holdsworth.

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CONGENITAL FUSION OF THE LUNATE AND TRIQUETRAL BONES IN THE SOUTH AFRICAN BANTU

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Congenital fusion of the lunate [semilunar] bone with the triquetral [cuneiform] is an abnormality which has been considered to be rare, even in negroes, who have been regarded as specially subject (Shanks 1950). Smitham (1948) estimated its incidence in a series of West African natives to be one in seventeen (5·9 per cent), and if his experience is typical it is certainly much commoner in some negro peoples than has been supposed. It is a very widespread abnormality, which has been described in American negroes (Paterson 1893, Derry 1906, Dwight 1907, Lönnerblad 1936, Alexander and Johnson 1941, Smitham 1948), in the Sudanese (Elliot Smith 1903, Lönnerblad 1936), and even in Australian aboriginals (Smith 1907).

The Bantu, with whom this paper is concerned, have this type of carpal fusion fairly frequently. They are a group of negro peoples who form a native population of southern Africa, to which they appear to have migrated from equatorial Africa. Their original habitat was probably the Nile Basin, which is believed to be the original home of all the negro races. The West African peoples, who migrated south of the Sahara Desert also from the Nile country, provided the ancestors of the American and West Indian negroes, and it is considered that even the Australian aboriginals can be traced back to the Nile, for the East Asiatic Papuo-Melanesians are a branch of the negroid division of mankind. It is, of course, to be expected that any abnormality which occurs in negro peoples would have a world-wide distribution.

It is not claimed that fusion of the lunate and triquetral are peculiar to the negro races. It has been described in white persons by Curr (1946), by McConnell (1907) and by Hammond (1947), and in several patients whose race and colour were not mentioned by the reporters.*

SIGNIFICANCE

The anthropological significance of this anomaly has been the subject of interesting speculation, especially by Smitham (1948) in his intensive study of it in West Africans. It may represent an advance towards specialisation of the hand, or it may signify an attempt to stabilise the post-axial border of the hand, in which case it is probably a primitive adaptation which has persisted. This latter is the favoured view, for in most cases in negroes the fusion is bilateral, yet the negro is seldom other than right-handed. Indeed, left-handed unilateral fusions appear to have been reported only in white people (Lönnerblad 1936, Curr 1946, Hammond 1947). It can be argued, perhaps a little teleologically, that any characteristic appearing to increase the stability of the weaker, post-axial border of the hand could well be usefully retained in the evolution of a race which has provided most of the world's slaves and whose members, slave or free, have had to fend for themselves with a minimum of even the simpler mechanical aids.

Lunate-triquetral fusion never causes symptoms. It is always found by accident. It may run in families (Mestern 1934). It does not appear to be associated with congenital anomalies in the feet.

* Three such cases were described by Lönnerblad (1936), two by Grashey (1917) and by Bogart (1932), and one each by Canigiani (1936), Reiss (1936), Belot and Nahan (1936), Lemarque and Betoulières (1939), McGoey (1943) and Hudson (1943).

TYPES OF FUSION

The writer has met twelve examples of carpal lunate-triquetral fusion in Bantu patients who were undergoing radiography for recent injuries. All were manual labourers. One was an amateur boxer of ability.

FIG. 1

Bilateral incomplete fusion of lunate and triquetral bones resembling pseudarthrosis.

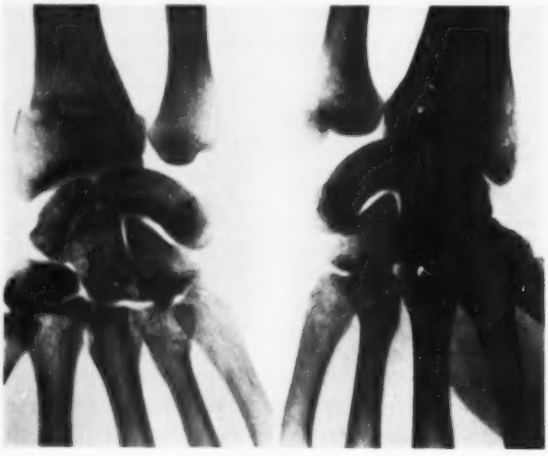

FIG. 2

FIG. 3

Figure 2—Bilateral fusion of lunate and triquetral bones with notch in distal border of the complex bone.
Figure 3—Fusion with notches in proximal and distal borders of complex bone. Right hand not radiographed.

The fusions were of four types: 1) *Incomplete fusion resembling a pseudarthrosis* in the radiograph. This type (Fig. 1) was found in both hands of a patient who had a fracture of the lower end of the right radius, but who had no other disability and who would admit to no other previous injury. The left wrist was normal in appearance and power. 2) *Fusion with a notch of varying depth* at the site of the usual division between the two bones. This notch

may be found at the distal contour of the compound bone (Fig. 2) or at both contours (Fig. 3). 3) *Complete fusion of lunate and triquetral alone* (Fig. 4). 4) *Complete fusion associated with other carpal anomalies.* This fourth type was seen only once in the present series (Fig. 5), in a wrist in which the hamate [unciform] was fused to the capitate bone [os magnum] and the

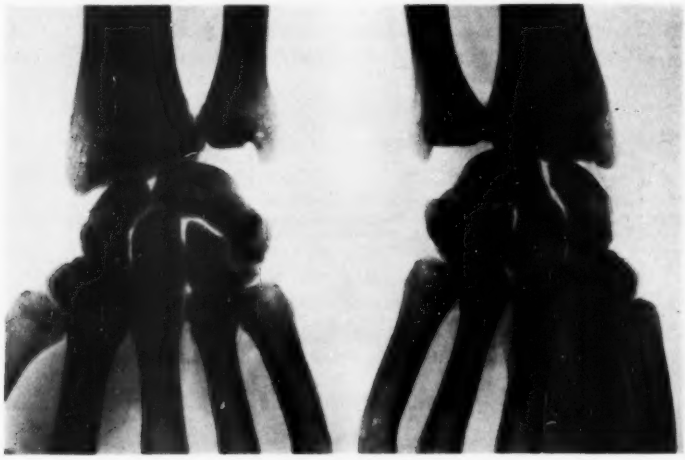

FIG. 4

Bilateral complete fusion of lunate and triquetral bones.

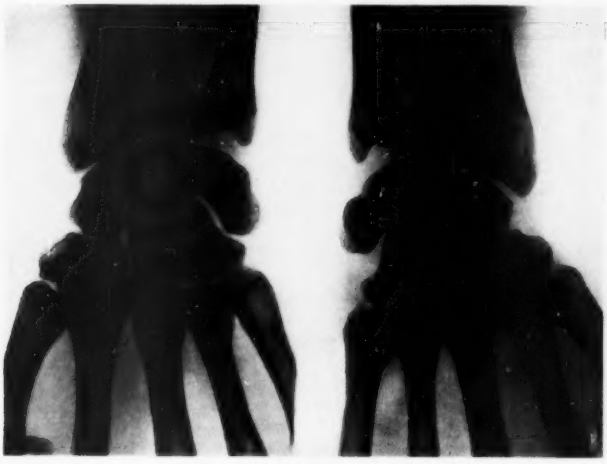

FIG. 5

Bilateral fusion of lunate and triquetral bones with bilateral capitate-hamate fusion and trapezium-trapezoid fusion.

trapezium [os multangulum majus] to the trapezoid [os multangulum minus]. The incidence of these four types and that of other carpal fusions has not yet been established. Bogart (1932) found three complete and three incomplete fusions of the capitate and the hamate in a series of 1,452 wrists, and in one of the latter three there was also fusion of the trapezoid and trapezium. These other fusions have been recorded by Dwight (1907)

and by Hudson (1943), but Smitham (1948), in a series of 680 wrists, found that in only one of six cases of hamate-capitate fusion was there a lunate-triquetral fusion as well.

SUMMARY

Twelve cases of congenital fusion of the lunate and triquetral bones have been found in South African Bantu patients. The fusion occurs in four types, an incomplete "pseudarthrosis," bony fusion with a dividing notch at one or both surfaces, or as a complete compound bone with or without other carpal anomalies.

The suggestion favoured is that this fusion represents the persistence of a primitive characteristic in African (negro) peoples.

I am indebted to Dr G. Findlay, Radiologist-in-Charge at the Baragwanath Hospital, and to his assistant radiologists for taking special radiographs. I am also indebted to the Surgeons-in-Charge for permission to use their cases, to Dr Maurice Weinbren for help with radiological interpretation and in finding references, and to Dr James Gilroy and Mr J. M. Edelstein for valuable assistance.

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TRANSFER OF THE TIBIALIS ANTERIOR TENDON FOR RELAPSED CLUB-FOOT

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In 1923 Dunn advocated transplantation of the tibialis anterior tendon in selected cases of congenital talipes equinovarus to guard against relapse, but he did not report his results. In 1940 Garceau reported the late results of the operation in forty-four patients (fifty-six feet). In 1947 Garceau and Manning made a study of the results in eighty-six patients and in 110 feet. They reported good results in 82·7 per cent of this series.

Whatever may be the initial cause of congenital talipes equinovarus, muscle imbalance is an important factor in the maintenance of the deformity once relapse has occurred. When deformity is severe it is obvious that the evertors and the dorsiflexors are stretched over the convexity of the dorsum and lateral border of the foot, whereas the tibialis anterior and

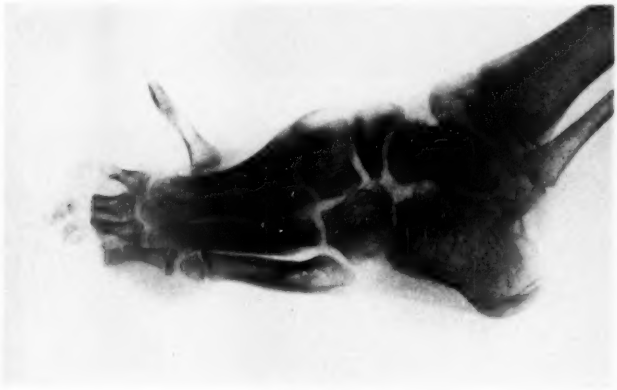

Fig. 1

Radiograph of the deformed foot in a child aged nine years. Well marked bony deformity is present, especially in the navicular and talus. Such a case is unsuitable for the operation described. If transplantation of the tibialis anterior is undertaken it should be done before secondary bone changes have occurred.

tibialis posterior extend in a straight line to their insertion in the concavity of the medial border of the foot. Not only do these muscles act at a great advantage, but the tibialis anterior acts almost as a pure invertor, with little power as a dorsiflexor of the foot. It should be remembered that all tendons, except the tendo calcaneus, are inserted in front of the midtarsal joint, and that the bone least affected in club-foot, namely the talus, receives no muscle attachment except a slip from the tendon of the tibialis posterior. When deformity recurs soon after adequate treatment is stopped, it develops so rapidly that it cannot be attributed to the contraction of fibrous tissue or to an anomaly of bone growth. It has been accepted in other conditions that the most rapidly deforming factor in a limb is muscle imbalance.

PRINCIPLE OF THE TIBIALIS ANTERIOR TRANSFER

Examination of patients with relapsed club-foot revealed a relative weakness of the evertors. There may be some correlation between the deformity and the fact that the peronei are the last muscles of the leg to receive their nerve supply (Harris 1942). It is also possible that intra-uterine pressure may damage the common peroneal nerve as it passes round the neck of the fibula, and thus lead to muscle imbalance. If the hypothesis of muscle imbalance is correct, lateral transfer of the insertion of the tibialis anterior tendon, after the deformity

FIG. 2

FIG. 3

FIG. 4

Case 1. Figures 2 to 4.—Typical congenital talipes equinovarus deformity before correction and lateral transplant of tibialis anterior tendon.

has been corrected, should prevent it from recurring, for the transplanted tendon will act as a dorsiflexor and as an evertor. Camera (1950) believes that transplantation of the tibialis anterior may lead to over-correction of the deformity, and he prefers to attach the two outer tendons of the extensor digitorum longus to the distal third of the fifth metatarsal. While not denying the value of Camera's method, we submit the present review as an indication of the efficacy of tibialis anterior transplants done in this hospital during the last four years. So far there has been no case of over-correction of the deformity after the transplant.

CLINICAL MATERIAL

The series includes nineteen patients. Operation was undertaken on both feet in three, making a total of twenty-two tendon transplants. Nine were done in the right foot, and thirteen in the left. Eleven patients were females, and eleven males. The age at the time of operation varied from three to thirteen years, with an average of five and a half years; and the follow-up period varied from six months to four years. Most of our patients had previously had operations such as plantar fasciotomy, or Steindler's operation, or elongation of the tendo calcaneus.

FIG. 5

FIG. 6

FIG. 7

Case 1. Figures 5 to 7—Condition four years after operation, showing improved appearance of the feet and active dorsiflexion and eversion.

TECHNIQUE OF OPERATION

The best age for operation is between three and six years. After the age of six years secondary changes in the soft tissues and bones prevent adequate correction (Fig. 1). It is well known that a tendon transplant will not correct a fixed deformity. For this reason over-correction by manipulation and plaster and if necessary elongation of the tendo calcaneus, with or without posterior capsulotomy of the talo-calcaneal and ankle joints, may be required to over-correct the deformity before transplantation.

At operation the tendon of the tibialis anterior is transplanted into the cuboid or, if the tendon is sufficiently long, into the base of the fifth metatarsal bone. An above-knee walking plaster, applied with the foot in the slightly over-corrected position, is worn for six months. After removal of the plaster a course of re-educative exercises is advised.

RESULTS

Results have been classified as good, fair, and bad. The criteria for a good result were: absence of pain, normal appearance, no limp, plantigrade position, active dorsiflexion and eversion (Figs. 2 to 7). These requirements were met in sixteen patients.

The criteria for a fair result were: absence of pain, plantigrade position, no limp and only slight residual deformity. There were two patients in this group.

Four patients have had bad results. Failure was attributed in one patient to rigidity of the feet caused by many previous operations. In another patient the tendon separated from its new insertion. There was persistent deformity in two patients, probably caused by too short a period of post-operative immobilisation.

We have observed that after transplantation of the tibialis anterior the extensor hallucis longus takes over some of the original function of the tibialis anterior: thus, during active dorsiflexion the hallux first hyperextends at the metatarso-phalangeal joint, then the foot dorsiflexes.

SUMMARY AND CONCLUSIONS

1. Twenty-two cases of transplantation of the tibialis anterior for relapsed club-foot have been reviewed. Results were good in sixteen cases, fair in two and bad in four.

2. The operation and routine treatment are described.

3. The reasons for the four failures are discussed.

4. It is concluded that the operation is well worth while, and will often avoid the need for bony stabilisation at a later date.

We are indebted to the late Mr G. R. Girdlestone, and to Mr W. B. Foley, Mr J. C. Scott and Mr E. W. Somerville for permission to investigate their case records. We wish especially to express our appreciation to Professor Trueta and to Mr J. C. Scott for their helpful criticisms and valuable assistance in the preparation of this paper.

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CHONDRO-OSTEO-DYSTROPHY

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Chondro-osteo-dystrophy is the name given by the author in 1928 to a disorder of the skeleton which manifested itself during the first three years of life. The child originally described had appeared to be normal at birth but at the age of five months the mother noticed that he had developed a kyphosis in the thoraco-lumbar area. At the age of eighteen months he was able to walk, but always appeared to take up a characteristic position at rest, the body being supported by the hands against the thighs, or, more frequently, by the hands on the flexed knees. In this position the thoraco-lumbar kyphosis was obvious and so was an apparent shortening of the neck. All the joints of the extremities were swollen and he developed knock-knees—appearances which led to the condition being diagnosed and treated as rickets. He looked and was bright and intelligent at three and a half years, when I took his first radiographs (Fig. 1).

Anti-rachitic treatment and thyroid medication were given over a long period but they met with no response. (So far no success has been achieved in any of the patients with any form of treatment.) A back support was fitted because of the kyphosis, which was thought to be due to tuberculous caries. Until the age of ten years he was able to run about even without this support, but it was noticed that he readily tired and took up his hand-knee attitude. This attitude he assumed also during sleep. From the age of ten years he steadily and progressively declined; the musculature became flaccid and atrophied as if in association with the general dystrophic condition of the skeleton. He did not grow, though his limbs appeared long in comparison with his trunk; his neck appeared to get shorter. Towards the end of his twelfth year of life his facial characters became grotesque and when he died at the age of fourteen, after being bedridden for nearly a year, he was but two feet ten inches tall and in weight just over two stone (28 lb., 13 kg.).

FIG. 1

Case 1—The subject of the author's original description at the age of three and a half years. He was then a bright and intelligent boy.

RADIOGRAPHIC EXAMINATION

The radiographic appearances of his skeleton at the time were unique. I had seen but two cases which bore any resemblance—a "forme fruste" chondrodysplasia observed by Silfverskiöld (1925) and a somewhat similar condition noted by Grudzinski (1928). Because the radiographs indicated defective and irregular ossification of the epiphyses and growing ends of the diaphyses, which appeared to have become manifest since birth, the term chondro-osteo-dystrophy was applied (after consultation with Sir Arthur Keith) and it was under this term that I demonstrated the radiographs at the Royal Society of Medicine in 1928. The distinctive radiographic features of the skeleton were illustrated and described (Brailsford 1929). At that time we had little serial radiographic evidence of the course of the condition and the purpose of this paper is to present such evidence gleaned from radiographs taken during sixteen or

more years of life in a number of similar cases (Figs. 2 to 21). Morquio described the clinical features of patients with this condition in 1929.

Early radiographic features—The chief radiographic features of chondro-osteo-dystrophy in the early stages are as follows. *Skull*—No appreciable change from the normal. *Spine*—The outlines of the bodies and processes are very irregular and ill-defined (Fig. 17). The bodies are flattened, some showing an anterior tongue with no ossification in the upper and lower epiphysial sites, some being slipper-shaped and of irregular size. The spinal curvature is altered; in the antero-posterior radiograph there is often a lateral displacement at the thoraco-lumbar junction. In the lateral, alteration in curvature may be seen in the cervico-thoracic and in the thoraco-lumbar areas (no epiphyses for the upper and lower plates have been seen even up to twenty-two years of age). In the cervico-thoracic area the compression is associated with shortening of the neck; in the thoraco-lumbar area a kyphosis has been seen associated with vertebral bodies irregular in size and shape; the disc spaces may be as deep as the flattened vertebral bodies. With increasing flattening of the vertebrae the sternum is pushed forwards progressively (Fig. 3). *Extremities*—The long bones have been of normal density and have not shown any unusual fragility. The epiphyses have multiple ossific areas and diaphysial extremities are irregular in ossification. The joint spaces, more particularly of the shoulder, wrist, hip, knee and ankle joint, are considerably widened, suggesting overgrowth of cartilage. The nuclei for the carpal and tarsal bones show delay in development and marked irregularity of outline (Fig. 12).

Later radiographic features—By a study of the serial radiographic appearances of the skeleton of a number of cases through life it was noted that while the skull remained relatively normal the epiphyses of the larger joints—notably the hip joints—deteriorated (Figs. 6 to 9). The ossific nuclei, which had originally been well formed, disappeared and no further ossification in them occurred. The musculature became progressively weaker, with the result that the joints became unstable and dislocation at the hip joints occurred. The acetabula expanded to the anterior spine and the upper end of the femora (which tend towards valgus deformity) came into cartilaginous apposition with the lateral aspect of the ilia; but later this connection was lost and the joints became somewhat flail—the opposing bony surfaces were irregular in outline being separated probably by dystrophic cartilage (Figs. 6 and 9). In one case subtrochanteric osteotomies were performed to counteract the dislocations, but these were unsuccessful in checking the displacement—indeed the operation appeared to accelerate the dystrophic disintegration (Figs. 14 and 15). At the knee joint, because the epiphyses are ossified from multiple nuclei which have not the stability of the normal entire homogenous nuclei, and because the ossification of the diaphysial extremities is irregular and mechanically unsound, knock-knee deformity develops without any bowing of the shafts of the diaphyses as in rickets (Fig. 13). In the forearm, however, the dystrophic changes at the wrist are associated with bowing due to irregular growth (Fig. 12). Osteotomy, which throws strain on dystrophic cartilage that is undergoing irregular ossification, appears to aggravate the process, but union occurs regularly through surgical fractures of consolidated bones (Fig. 13).

ETIOLOGY

It seems probable, from examination of the cases which have come to me since the original presentation, that the condition occurs in both sexes and is associated with some lethal factor in the parents introducing a state of incompatibility. The gravity of the lethal factor appears to be accentuated by consanguinity of parentage; this is illustrated in the case I published of a dystrophic child, the offspring of a brother and sister aged sixteen and fourteen years respectively. But we cannot entirely rule out immaturity of parents or the employment of abortifacients in this case. Disparity in the ages of parents has not been present where all of the children have shown these dystrophic changes in a grave form and no defect has been found in the parents. In one family which had three children so affected,

FIG. 2

FIG. 3

Case 2. Figure 2—Photographed with a normal youth of the same age, eighteen and a half years. Figure 3—Detail photographs showing dwarfing, deformity of the sternum, enlargement of the joints of the extremities, and displaced hip joints. (See also Figures 6 to 12.)

FIG. 4

FIG. 5

Figure 4—Cases 4 and 5. The mother with two affected children. The boy (right, Case 4) is aged ten years, and his sister (Case 5) is aged twenty-two years. The girl is mentally advanced for her years. Figure 5—Case 6. Girl aged thirteen and a half years photographed with her brother aged ten years. The girl is now aged twenty-two years. She has not grown since the photograph was taken but is mentally advanced for her years.

FIG. 6

FIG. 7

Case 2. Figure 6—Pelvis and hip joints at the age of fourteen months. Note well developed regular epiphyses; some increase in joint space, and coxa valga. Figure 7—Condition at age of four years and eight months. Note increased joint spaces and deterioration in epiphyses and walls of acetabula. Coxa valga deformity.

one died at two years of age; the others are ten and twenty-two respectively. The eldest child, a youth of twenty-three years, has a further complication associated with fits. The father did not walk until seven years of age, though of normal stature; he died two years ago of aplastic anaemia.

In such cases the lethal factor responsible (which might possibly be the employment of some abortifacient; more widely used perhaps than generally known) operates with each pregnancy but the gravity of the condition has shown some variation. Though syphilis is a common cause of developmental defects, in these patients its presence has not been detected. It has been shown experimentally that irregularities in development can be produced by agents which result in checking growth, interfering with oxygenation, or in the production of toxic products. The effect of these is dependent upon the age of the embryo, the rate of growth of the respective tissues at the time the agents are active, and the duration of time

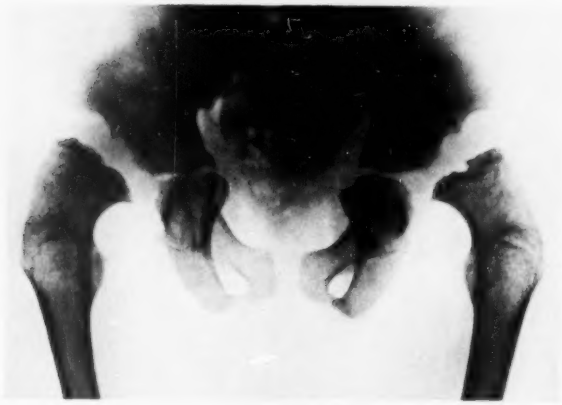

FIG. 8

FIG. 9

Case 2. Figure 8—Condition at age of fourteen years ten months. Note further degeneration of epiphyses and acetabular walls, increased joint space and dislocation. Figure 9—Condition at eighteen and a half years. Note complete dislocation and marked irregularity of all " articular " surfaces.

during which the agents are applied. Similar deformities are produced by different agents acting at the same stage of development. It has not been possible experimentally, even with the earliest implantations, to determine with accuracy the relative contribution of heredity and environment, and though the familiar distribution tends to support the possibility of hereditary lethal factors in the parents we cannot exclude the presence of deleterious agents, toxins—infective or metabolic—or abortifacients, in environment.

In all cases there appears to be induced an associated constitutional weakness, so great in some as to be incompatible with separate life; in others, less severe, the infant survives a few years and then dies from pneumonia or, as in the case of my original patient who died at fourteen years, from a progressive degeneration of all the skeletal tissues. In others—as in the cases which I have now watched for nearly sixteen years and whose radiographs are presented in this paper—the dystrophy shows progressive development and results in dwarfing

FIG. 10

FIG. 11

Case 2—Spine showing, in the antero-posterior view (Fig. 10) irregular disposition of the vertebrae and, in the lateral view (Fig. 11), "slipper" form of the vertebral bodies, absence of vertebral epiphyses, and disc spaces as wide as the vertebral bodies.

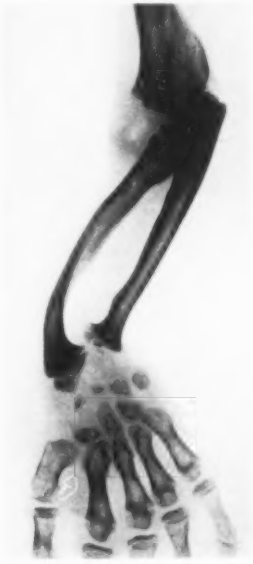

FIG. 12

FIG. 13

Case 2. Figure 12—Elbow, forearm and wrist at the age of nineteen and a half years. Note irregularity of growth at the elbow, bowing of radius and ulna with delayed ossification, irregular and delayed growth of carpal bones and irregularity of base of metacarpals. Figure 13—Tibia and fibula at the age of eighteen years. Note irregularity and obliquity of joint surfaces and site of osteotomy performed five years previously.

of the skeleton and weakness of the musculature; nevertheless the patient survives to adult life, is of normal or perhaps supernormal intelligence and highly sensitive of the dwarfing, resenting with very reasonable argument any unnecessary investigation or demonstration.

FIG. 14

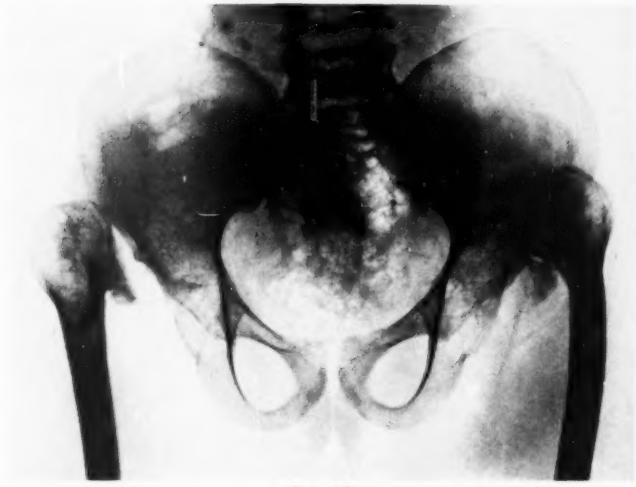

FIG. 15

Case 3. Figure 14—Hip joints at the age of three and a half years. Note failure of ossification of femoral necks and epiphyses. Figure 15—Condition at the age of twenty-four years. Bilateral subtrochanteric osteotomies were performed at the age of seventeen years. Compare position with that shown in Figure 14.

I have watched by periodic radiographic and clinical examinations for over sixteen years the sequence of changes in the members of several families with this dystrophy. They are so alike in all their clinical and radiographic features as to be indistinguishable from one

FIG. 16

FIG. 17

Case 4—Spine and pelvis at the age of ten years. In the antero-posterior view (Fig. 16) note the irregular disposition of the vertebrae and dislocation of both hip joints. The lateral view (Fig. 17) shows "tongue" shaped thoracic vertebral bodies and irregularly sized "shipper" shaped upper lumbar bodies. The disc spaces are wide.

FIG. 18

FIG. 19

Figure 18—Case 4. Hands at the age of ten years showing irregularity and delay in ossification of carpal bones and growing extremities of long bones.
Figure 19—Case 5. Left hand at the age of twenty-two years showing that the epiphyses have fused but the joint surfaces are irregular and some of the bones stunted in growth.

another at certain stages. They constitute a distinct entity. Their ability to get employment is dependent upon the extent of their physical endurance—muscular weakness, headache and eyestrain may seriously curtail activities. Examples of mild forms of the dystrophy have

FIG. 20

Case 5—Pelvis and hip joints at the age of twenty-two years showing dislocation of both hip joints, marked expansion of the acetabula, and wide and irregular symphysis.

FIG. 21

Hip joints of a patient, otherwise normal, showing disturbed ossification of the head and neck of the right femur caused by injury.

been encountered in the examination of adult dwarfs, who have had no other handicap than the dwarfing, in an otherwise normal life (Engel 1938). They have sometimes been mistaken for achondroplasiacs but they have the distinctive features of a relatively short trunk compared

with the limbs. Cases show not only variation in gravity of the generalised condition but variation in the degree of disturbance in different joints—for instance, the changes may be extensive in the hip joints or spine with little irregularity in the other joints.

DIFFERENTIAL DIAGNOSIS

Rickets—Florid rickets in infancy is characterised by generalised osteoporosis in which the outlines of all the diaphysial extremities and of the early ossific nuclei for the epiphyses are blurred (Brailsford 1948). The epiphyses are ossified from one central nucleus. The bones show absence of compact tissue, exhibit plasticity, and bend with weight-bearing; the deformities of the skeleton, being due to this, are symmetrical and generalised. Since the bones are deficient in calcium they fracture more readily than normal; some show multiple so-called pseudofractures. Consolidation of the bones promptly follows administration of adequate doses of vitamin D and no further deformity develops—indeed considerable correction may occur with growth. More rarely there are cases which show resistance to vitamin D and other medication or are associated with renal disease or hyperparathyroidism, the characteristic features of which are described elsewhere.

Hypothyroidism—In this condition there is generalised delay in ossification and the epiphyses are late in showing ossific nuclei; when these do appear they are multiple, and, because the patient is fit enough to subject the epiphyses to weight-bearing and normal function, they later become deformed, for they have not the stability of the epiphysis formed from homogeneous central ossification. The large epiphyses, such as the femoral capital epiphyses, show these defective features best. Good response, with acceleration in growth, consolidation and fusion of the multiple ossific nuclei, follows adequate thyroid medication (Brailsford 1948).

Osteochondritis—Although several epiphyses may simultaneously exhibit the characteristic series of changes which follow avascular necrosis, it is more common to find only isolated epiphyses affected. Sometimes both femoral capital epiphyses are affected, but even in these it may be possible to recognise that the change was initiated in each at a different time. Deformities are due to pressure during the plastic stage. Injury to a young epiphysis may delay the appearance of the ossific nucleus, or may cause its decalcification and deformity and result in a delayed ossification from multiple nuclei and permanent deformity (Fig. 21). Such deformity associated with stunted growth may result from the trauma inflicted during reduction of a congenital dislocation.

Achondroplasia—This condition is distinguished as a fault in the architect's plan—the long bones and the bones at the base of the skull are prematurely fused, shortened and thickened, and the bones of the pelvis are squat. The vertebral bodies tend to be square in shape and at birth there is an unusual localised regular thoraco-lumbar kyphosis without any change in the form of the bodies; the epiphysial growth is from single nuclei which progress regularly to maturity (Brailsford 1948). The tuberosities show unusual development. The constitution is good and the musculature well developed, whereas in chondro-osteo-dystrophy the skeletal plan as seen in the newly born may be normal, the defects being due to faulty building material. Occasionally the achondroplasiac (even as the normal) may show the additional features of chondro-osteo-dystrophy. In other words hereditary developmental irregularities do not render the skeleton immune from dystrophies, any more than from deficiency disease. Some irregularities of the skeleton appear to be due to their mixing. The features of achondroplasia and chondro-osteo-dystrophy have been well illustrated by Fairbank (1949).

Gargoylism—There is a miscellaneous group of dwarfs who are grotesque in appearance. They may or may not exhibit generalised irregularities in the growth of the skeleton which produce dwarfism; there may also be defective development of other systems such as the nervous, circulatory, respiratory, alimentary, genito-urinary or endocrine systems in various combinations. The possible combinations indicate the difficulty in any attempt at their classification. Gargoylism should be regarded as a general term, it cannot be said to include

any particular character other than a grotesque appearance; even the skeletal lesions may be widely dissimilar. The grotesque dwarf whose features I illustrated and described (Brailsford 1943) had skeletal characters entirely different from those of chondro-osteo-dystrophy or any other case described under the term gargoylism, but the grotesque appearances were similar.

SUMMARY

1. The evolution of the skeletal changes in chondro-osteo-dystrophy is described, and typical radiographs of patients observed into late adolescence are shown.
2. The etiology of the condition is discussed.

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TRAUMATIC CHYLOTHORAX

A Case After Dislocation of the Thoracic Spine

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From the Accident Service and Department of Pathology, Radcliffe Infirmary, Oxford

Several cases of traumatic chylothorax have been reported, almost all of them in American papers. Baldridge and Lewis (1948) and Lampson (1948) published a review of the literature and described the clinical course and methods of treatment. They point out that in earlier years crushing injuries of the chest and stab and bullet wounds were responsible for most of the thoracic-duct injuries (Shackelford and Fisher 1938), but recently many have occurred during intrathoracic operations.

CASE REPORT

A lorry driver aged twenty-seven years was admitted to another hospital after a road accident in which he had been crushed inside the cab of his lorry. As far as could be found, his head and shoulders had been forcibly flexed over the steering wheel by the collapse of the cab roof. On admission, he was conscious but severely shocked. His lower limbs were paralysed and he had loss of sensation which indicated complete interruption of the spinal cord in the mid-thoracic region; there were also multiple fractures of the mandible. Radiographs taken after resuscitation showed complete dislocation of the sixth on the seventh thoracic vertebrae (Fig. 1). It was thought that the chance of recovery of the spinal cord lesion was too slight to justify any attempt at reduction of the displacement while his general condition remained poor. Resuscitation was continued, and on the following day he was transferred to the Accident Service, Radcliffe Infirmary, Oxford, with the dislocation unreduced.

Two days after injury his general condition was much improved, but he had severe abdominal distension and absent bowel sounds, and he was vomiting much dark brown fluid. Rales were heard in both lungs. Operation was therefore again postponed. A radiograph of the chest at this time showed a small quantity of fluid in both pleural cavities, but no pulmonary collapse. The next day the abdominal distension was less severe, the bowel sounds were audible, and vomiting had ceased; operation was then undertaken. The mandibular fractures were reduced and immobilised by wiring of the jaws (Mr D. S. Hayton Williams). Manipulative reduction of the spinal dislocation was performed (Mr R. G. Taylor), and the spine was immobilised in a plaster jacket because of the need for postural drainage of the respiratory tract. The patient's general condition was satisfactory, and it remained so for the next two days. He was able to drink about five and a half pints a day, but his output of urine remained low, at thirty-five to forty ounces a day. The blood pressure, pulse, temperature and respiratory rate were steady, and abdominal distension did not recur. Clinical examination and radiography of the chest were prevented by the plaster jacket. Four days after operation and one and a half hours after a meal he became suddenly distressed and cyanosed, lost consciousness, and died within ten minutes.

Necropsy—There was complete dislocation of the thoracic spine between the sixth and seventh vertebrae, with an associated transverse fracture of the sternum two inches above the xiphoid. The spinal cord had been completely transected at the site of the dislocation, though a bridge of dura mater remained. For one inch above and below the cord injury there was a haematorrhachis, and four inches below the lesion at the site of the lumbar enlargement there were multiple petechiae and small haemorrhages in both the white and

grey matter. Parts of the intervertebral disc at the site of the dislocation projected through large tears in the mediastinal pleura into both pleural cavities. The oesophagus was intact. There was a pleural effusion of 1·5 litres on each side, the fluid of which resembled thin white coffee, but was tinged with red. It contained some fat and 1 gramme per cent of protein; it was still opaque after it had been centrifuged, and the deposit consisted of red cells and many lymphocytes, with a few macrophages and endothelial cells. Thus the chylous nature of the fluid was certain, but unfortunately its source was not demonstrated and it must be presumed that there had been severe injury to the thoracic duct at the site of the spinal dislocation. There was almost complete collapse of both lungs, with contusions posteriorly at

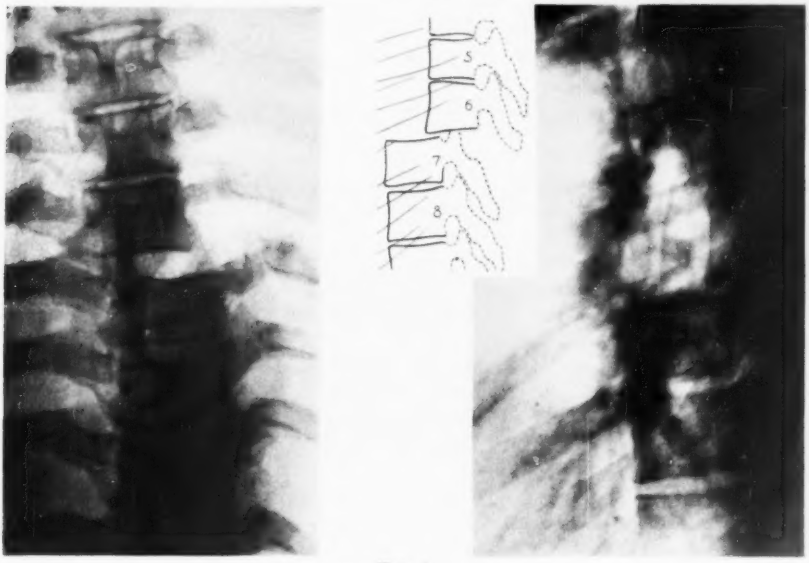

FIG. 1

Radiographs of the thoracic spine taken on admission. The anterior surface of the body of the sixth vertebra lies almost in line with the posterior surface of the body of the seventh.

both apical and hilar regions. The right side of the heart was dilated, and the left side and aorta were empty: subendocardial haemorrhages were prominent in the atrioventricular [auriculo-ventricular] regions, and an ante-mortem thrombus was firmly attached to the atrial [interauricular] septum near the fossa ovalis in the right atrium. There was no pulmonary embolus. The liver and kidneys were congested and petechiae were scattered through the brain.

DISCUSSION

The normal flow of chyle in the thoracic duct is between sixty and 190 millilitres an hour, depending on several factors—particularly the intake of food and water. The chyle is responsible for the transport of 70 per cent of ingested fat from the intestine to the blood; it also plays a large part in the transport of protein (Co Tui *et al.* 1944), which may be present in a concentration as high as 8·0 grammes in 100 millilitres. The cells are predominantly lymphocytes; obstruction of the thoracic duct causes a fall in the number of circulating lymphocytes and eosinophils, but no adequate explanation of the diminution of eosinophils has been suggested.

Three to four days usually elapse after injury before the onset of symptoms of chylothorax. It has been assumed that the chyle at first accumulates in the posterior mediastinum, and it

is only after several days that the pleura on one or both sides gives way and allows a sudden leakage of fluid into the pleural cavity. Symptoms then appear suddenly and are at first referable only to the mechanical effects of the large pleural effusion; there is striking relief of these symptoms after thoracentesis, but they recur as the effusion collects again. Should leakage continue for many days, a second group of symptoms appears due to the loss of fat, protein and fluid. The patient rapidly loses weight and becomes dehydrated; there is a reduction in serum protein and a fall in the number of circulating lymphocytes and eosinophils which if unchecked causes death.

Except in cases where chylothorax has resulted from damage to the cervical part of the thoracic duct treatment has been almost entirely conservative, because of the high mortality associated with attempted surgical relief of intrathoracic ruptures. The results of conservative treatment have been far from encouraging, however, and most authors record a mortality of between 40 per cent and 50 per cent (Loe 1946). Conservative treatment has three objects: to relieve the pressure effects of the pleural effusion, to maintain nutrition, and to encourage spontaneous closure of the ruptured duct. The first is attained by repeated aspiration of the pleural cavity, restriction of fluid and fat by mouth, and maintenance of the normal plasma-protein level to restrict capillary transudation and the formation of lymph. Measures to maintain nutrition have included a high protein diet, transfusion of blood and plasma, and in a few cases intravenous transfusion of the chyle aspirated from the chest (Bauersfeld 1937). The treatment most likely to encourage closure of the duct fistula has been the subject of much discussion. Artificial pneumothorax, phrenic nerve interruption and pneumoperitoneum have all been tried, but recently interest has increased in the possibility of a more direct approach.

Many investigators have shown that the thoracic duct can be ligated without ill-effect. Blalock (1937), working with dogs and cats, showed that it is difficult to prevent the discharge of lymph into the circulation by ligature of the thoracic duct and its branches. Recently several cases of successful intrathoracic ligature of the duct in man have been described (Shumacker and Moore 1951).

The interest of the present case lies firstly in the spinal injury, which was an unusual one: no other instance of complete dislocation of the thoracic spine has been found in the records of the Accident Service of the Radcliffe Infirmary for the past eleven years. Secondly, the pleura on both sides must have been torn at the time of the injury, with the protrusion of intervertebral disc material into both pleural cavities. It was therefore possible for lymph to enter the pleural cavities from the beginning. Nevertheless, a radiograph forty-eight hours after injury showed so little intrapleural fluid that chyle formation must have been greatly restricted during this period; there was the usual delay between injury and the onset of symptoms, which cannot be explained by the accumulation of lymph in the mediastinum. If a normal and steady rate of flow of chyle be assumed, the quantity of lymph found in both cavities at necropsy would have taken about twenty-four hours to collect, during which time a gradual increase of respiratory embarrassment might have been expected. It is reasonable, therefore, to suppose that chyle did not flow at a steady rate, but that a sudden increase in flow took place immediately before death. This may have been due to: 1) haemoconcentration, increased osmotic pressure of the plasma proteins diminishing lymph formation; 2) ingestion of fluid and food, which increases lymph flow in the thoracic duct (Loe 1946); 3) anoxia, which by increasing the permeability of capillary endothelium augments lymph formation (Drinker and Yoffey 1941); 4) increase in venous pressure; this increases lymph flow in the thoracic duct as much as five times (Starling 1941).

During the first four days after injury haemoconcentration must have occurred, the intake of fluid and food by mouth was severely restricted, and it seems probable that lymph formation was correspondingly reduced; on the fifth day, however, improvement in the patient's general condition allowed an intake approaching normal, and the rate of lymph

production presumably increased to normal. The pleural effusions now accumulated rapidly and caused pulmonary collapse with a consequent rise in venous pressure. This in turn, with anoxia after the collapse, caused a further increase in lymph flow leading to fatal respiratory obstruction. There was evidence at necropsy that the venous pressure had been raised, as the right side of the heart was engorged and the left side was empty. The kidneys and liver were congested. A " vicious circle," initiated by an increased food and fluid intake, seems to provide a likely explanation of the delayed but sudden collapse in this case, and may well apply to other cases of traumatic chylothorax. Frequent examination to exclude pleural effusion after crushing injuries of the chest and injuries of the thoracic spine may result in diagnosis before the onset of pressure symptoms.

SUMMARY

A case is described of bilateral traumatic chylothorax after complete dislocation of the thoracic spine. An explanation is advanced for the delayed, sudden onset of symptoms due to the chylothorax in this and other cases.

We wish to thank Mr R. G. Taylor, under whose care the patient was admitted to the Radcliffe Infirmary, for permission to report this case; also Mr J. C. Scott and Dr A. H. T. Robb-Smith for advice and criticism in the writing of the paper.

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FRACTURE OF THE PELVIS COMPLICATED BY ISCHAEMIA OF THE LOWER LIMB

Report of a Case

J. N. Wilson, Oswestry, England

From the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry

A quarryman aged thirty-seven years was crushed between two wagons in May 1951. When admitted to hospital he complained of pain in the pelvis, with weakness and numbness of the right lower limb. He was found to have sustained a fracture of both pubic rami on the right side with separation of the symphysis and subluxation of the right sacroiliac joint (Fig. 1). His general condition was rather worse than one would expect from a fracture of the pelvis alone, but there were no signs of damage to bladder or urethra, nor of any other

Fig. 1

Radiograph showing separation of the symphysis pubis and fracture of the inferior ramus of the pubis on the right side. The fracture of the superior pubic ramus, found at necropsy, is not discernible.

severe injury. He had, however, sustained considerable local bruising, and it was thought that a large swelling in the right groin was probably a retroperitoneal haematoma. Sensation in the right lower limb was impaired, and there was general weakness in all muscle groups. There was no suggestion at that time of any circulatory disturbance.

His condition improved after admission and he was put up in a pelvic sling in an attempt to correct the separation of the symphysis pubis. About twelve hours later his general condition deteriorated fairly suddenly. The blood pressure dropped to 90 millimetres Hg. (systolic), and it was thought that further bleeding must have occurred into a retroperitoneal haematoma. Two pints of blood were given, with good effect, and the pelvic sling was

tightened. He soon complained, however, of pain and numbness of the whole of the right lower limb.

On examination, there was found to be no voluntary movement in the limb, and there was complete loss of sensation up to the groin; the ankle was pale and without apparent circulation, and the pulses were impalpable from the femoral downwards. The muscles of the thigh and calf were sharply outlined as they stood out in spasm, the foot being held in rigid equinus. A diagnosis of ischaemia of the right lower limb, probably due to spasm of the femoral artery associated with pressure on the main vessels by an increasing retroperitoneal haematoma, was made. It was thought that the only hope of preserving the limb was to explore the vessels and release the haematoma. But the patient's condition never recovered sufficiently to justify operation, and he died about twelve hours later.

FIG. 2

Part of the femoral artery overlying the right superior pubic ramus. The damaged part of the vessel is shown clearly in the centre of the specimen.

Post-mortem findings—The cause of death was found to be cerebral fat embolism. A large extraperitoneal haematoma was found on the right side extending down under the inguinal ligament, and lying over the femoral vessels. The femoral artery was narrowed in the section overlying the pubic ramus and contained ante-mortem clot in the area of constriction (Fig. 2). The damaged vessel was in immediate relation to a linear fracture of the superior ramus of the pubis, one edge of which impinged against the artery; this edge became more obvious on moving the pelvis. This fracture was not discernible in the radiograph of the pelvis. The muscles of the thigh and calf were pale in comparison with the other side, and the macroscopic appearance was one of ischaemia.

Comment—This case illustrates three points in relation to femoral artery damage in pelvic injuries. Firstly, how a fracture without evident displacement may damage the artery because of its proximity to the bone at this level. Secondly, the importance of the large retroperitoneal haematoma which may produce local pressure and increase the effect of any spasm already present. Thirdly, it is possible that tightening the pelvic sling may have caused some alteration in the fracture of the pubic ramus, for it was observed that the main symptoms of ischaemia began after the sling had been tightened.

I am indebted to Mr F. G. Allan for permission to publish this case.

COMBINED INJURY OF THE POPLITEAL AND POSTERIOR TIBIAL ARTERIES

Report of a Case

N. W. Nisbet, Dunedin, New Zealand

In February 1950 a printer aged nineteen years accidentally set in motion a printing press and crushed his right lower limb between a cylinder and a plunger. The limb was drawn into the cylinder as far as the lower part of the thigh. He was released immediately, and admitted to Dunedin Hospital.

Examination on admittance—The patient was severely shocked. The right lower limb was tightly swollen and there were abrasions over the popliteal fossa and leg. The knee was distended with fluid. Radiological examination showed a subcondylar fracture of the

Fig. 1

Subcondylar fracture of the tibia. The level of the fracture at the posterior aspect of the tibia is at the bifurcation of the tibial vessels.

tibia without displacement (Fig. 1). The junior hospital staff did not notice any sign of a serious vascular injury because the swelling made examination difficult; there can be little doubt that it would otherwise have been seen. The patient was treated for shock and the limb was lightly splinted.

Progress—The patient continued to be distressed and suffered severe pain. The swelling of the lower limb increased, and it was noticed that the toes and foot had no movement, were

analgesic and were becoming gangrenous. The patient felt that his foot was alternately hot and cold. It was then realised that there had been a severe vascular injury, and after taking into account the radiographic appearances it was thought that the tibial vessels had been injured at the site of the fracture. The whole foot rapidly became gangrenous, however, and an injury to the main popliteal artery was suspected.

As there was clearly no object in waiting, the limb was amputated through the thigh, making a ten-inch femoral stump. At the time of amputation it was noticed that the collateral circulation in the muscles was poor. The stump healed well, and the patient was allowed home three weeks later. A satisfactory limb was fitted after about three months and he returned to work.

FIG. 2

The dissected limb showing rupture of the popliteal artery. The bifurcation of the artery has been gripped and drawn forwards by the fracture of the tibia.

Morbid anatomy—The specimen was dissected by Dr Rodda of the Department of Pathology of the Otago University. Serious vascular injuries were found at two levels. 1) Complete rupture of the popliteal artery in the popliteal fossa. There was barely a drop of blood in the fossa, as the stumps of the artery had inverted and rapidly sealed themselves off (Fig. 2). 2) The posterior tibial artery had been drawn forwards over the crescentic margin of the interosseous membrane and with some of the surrounding tissues was tucked into the fracture of the tibia. The anterior tibial artery was represented by a narrow insignificant vessel. Apart from a little bruising, the veins and nerves were undamaged.

DISCUSSION

It is interesting to speculate on the mechanism of this injury. When the limb was drawn into the cylinder, presumably the fracture of the tibia occurred first and gripped the posterior tibial artery. This would put longitudinal tension on the main artery, which snapped like a bow-string when it became compressed in its turn.

DISLOCATION OF THE SHOULDER COMPLICATED BY RUPTURE OF THE AXILLARY VESSELS

Report of a Case

JAMES R. KIRKER, AUCKLAND, NEW ZEALAND

In July 1950 a man was admitted to hospital with the history that three weeks before he had dislocated his left shoulder joint. The dislocation had been reduced easily under thiopentone anaesthesia one and a half hours after the injury, and he had returned home. He suffered no discomfort that night, but forty-eight hours later the arm was greatly swollen and he had paroxysmal burning pain throughout the limb. There was no discolouration. The arm had not been examined for injury to the nerves or vessels before or after reduction of the dislocation. One week before admission, when the man was seen as an out-patient, he still complained of pain. On examination, he had extensive and unusual analgesia of the limb and paralysis of all muscle groups except the flexors of the finger. It was thought that he had a contusion of the brachial plexus or a traction injury, and the arterial pulses were again not examined. He was given a cock-up splint and sent to the physiotherapy department for muscle reaction tests and exercises.

Condition on admission—Examination suggested that there was a complete brachial plexus paralysis and an arterial vascular injury. *Left shoulder*—The bones were in normal relationship but no active movement was possible and slight passive movement only was tolerated. There was gross swelling in the supraclavicular and infraclavicular regions and a tense fluctuant mass was palpable beneath the anterior fibres of the deltoid, the pectoralis major and in the base of the axilla. *Left arm*—There was flaccid paresis of all muscle groups, including the supraspinatus and deltoid. Sensation was normal over the deltoid muscle but was impaired elsewhere, and discrimination was absent—that is, sensory impairment was present from the sixth cervical to first thoracic segments. The left subclavian pulse was half the volume of the right, and the brachial and radial pulses were absent. There was swelling of the whole arm with oedema and induration in the bicipital groove; a systolic bruit was heard over the swelling around the shoulder joint.

Two days later the radial pulse was palpable, but there was no brachial pulse. *Electrical reactions*—There was reaction of degeneration in all groups except the biceps muscle.

An arteriogram was made after injection of opaque fluid into the third part of the subclavian artery. This showed a normal brachial artery and a normal arterial system distal to it, but the axillary artery was not filled (probably because of the rapid blood flow). It was decided to observe the limb, and the only treatment given was daily passive movements to all joints.

Progress—Ten days later there was no improvement in the paresis and the swelling had not altered in size. A tentative diagnosis of axillary haematoma was made.

OPERATION

The axilla was explored by a delto-pectoral approach. The axillary space contained about 30 ounces of old blood clot under tension. There was a tear at the junction of the second and third parts of the axillary artery, which bled profusely as soon as the clots were removed; there was also a linear tear in the axillary vein. The medial and lateral cords of the brachial plexus were displaced downwards and forwards by the haematoma; there was

no anatomical disruption of the plexus. The third part of the subclavian artery was ligated and the tear in the axillary vein was sutured. Later on the same day the collateral circulation was adequate, the hand was warm and had good capillary circulation, but there was no radial or brachial pulse and no return of sensation.

POST-OPERATIVE COURSE

Four weeks after operation there was active power in the deltoid muscle but no other improvement in the neurological signs, and the radial pulse was still impalpable. Six months after operation there was return of sensation to the level of the wrist joint, useful flexion of the fingers and thumb, but no extension of the wrist or fingers. The pronators of the forearm were functioning and there was a flicker of movement in the brachialis.

Eight months after operation sensation to touch and pinprick had returned to the level of the proximal interphalangeal joints on the dorsum and palmar surface of the hand. Sensory discrimination was poor. In the shoulder, active abduction to 80 degrees was possible against resistance. The elbow could be flexed against resistance and extended against gravity; the range of elbow movement was 45 to 115 degrees. Pronation was full and supination was possible to the mid-position. Flexion of the fingers and thumb was full and powerful, but there was no active extension of the wrist or fingers. The intrinsic muscles were paralysed.

Comment—Useful sensation may return to the pulps of the fingers, but it is unlikely that the extensors of the wrist and fingers or the intrinsic muscles will recover. Arthrodesis of the wrist might be carried out later, and might be followed by a flexor sublimis transplant to the extensor expansions to the fingers. This would give useful function to the upper limb.

SUMMARY

1. A case is reported of traumatic dislocation of the shoulder joint complicated by rupture of the axillary artery and vein and complete brachial plexus palsy. Whether the rupture occurred at the time of injury or during reduction is not known.

2. Despite a delay of five weeks before operative treatment was undertaken the usefulness of the arm was preserved.

3. After rupture of the axillary artery at this level an adequate collateral circulation is rapidly established in a healthy young adult.

4. The case illustrates the importance of careful examination of the peripheral nervous and vascular systems before and after reduction of a dislocated shoulder.

My thanks are due to Mr Selwyn Morris, under whose care the patient was admitted, for permission to publish this account.

FIBULAR TRANSPLANT FOR OSTEOCLASTOMA OF THE RADIUS

T. L. Lawson, Sheffield, England

An African policeman aged twenty-two years came to hospital with the history that he had suffered a blow on his left forearm two months previously. There was a diffuse smooth swelling of mixed consistency over the distal quarter of his radius; the skin over it was red but not adherent, and movement of the wrist joint was partly impaired in all directions. Radiological examination showed a typical osteoclastoma of the lower end of the radius (Fig. 1) and this was confirmed by a microscopical examination after operation.

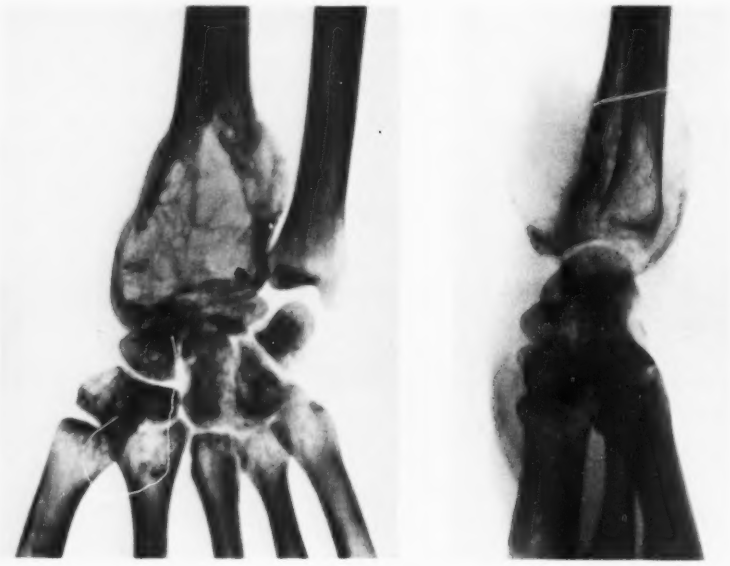

Fig. 1

Radiographs showing the osteoclastoma.

Operation—The lower end of the radius was excised well above the site of the tumour and a graft consisting of the upper end of his left fibula was inserted and fixed with vitallium screws. Figure 2 demonstrates the remarkable similarity between the fibula and the radius.

Progress—Convalescence was uneventful except for a large haematoma in the donor site. Ten days after operation the padded plaster was replaced by a skin-tight one, and active exercises were started. Three months later there was solid union, and the plaster was removed; the patient had a good grip, but only about 10 per cent of movement in all directions at the wrist. Three months after that, however, it was difficult to tell the difference between the gripping powers of the two hands and there was 65 degrees of movement in flexion and extension (Fig. 3). He then returned to duty, which included parades with a service rifle.

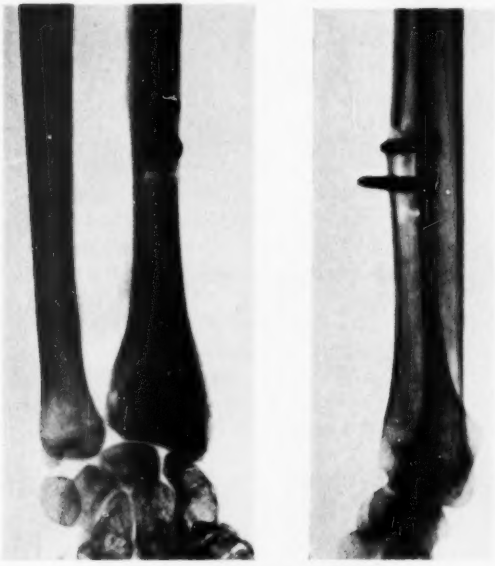

FIG. 2
The fibular graft in place after operation.

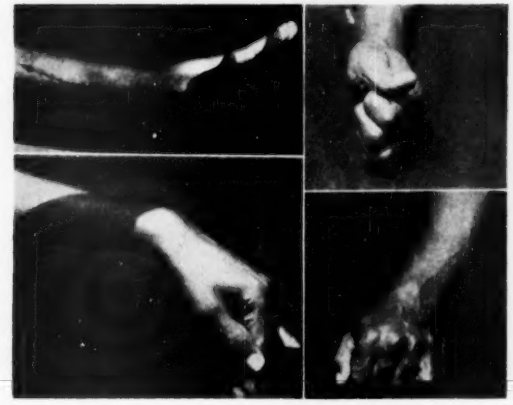

FIG. 3
Movement at the wrist after operation.

Comment—The patient's condition is now satisfactory a year after operation, but cases have been recorded in which massive grafts implanted after resection for osteoclastoma have themselves become the site for a similar tumour, though the excision has been wide. In this case there was undoubtedly some contamination of the wound with tumour cells at operation, but no recurrence has yet appeared.

JOINT CHANGES IN NEUROFIBROMATOSIS

Report of Two Cases

A. C. Bingold, London, England

Gould (1918) stated that neurofibromatosis may cause pigmentation of the skin, mental deficiency, changes in the skeleton and changes in the joints. What the joint changes were he failed to specify, nor have they been described by a later writer. I intend therefore to analyse the joint changes in neurofibromatosis, after presenting two illustrative cases.

CASE REPORTS

Case 1—A thirty-seven-year-old woman complained of intermittent pain in her left knee for the last twenty years, and of constant pain in her left groin for the last seven months. For as long as she could remember she had had lumps in her skin and a limp. She was the youngest of ten children; no other member of her family was similarly affected.

Fig. 1

Fig. 2

Case 1. Figure 1—Photograph of the patient's back showing many subcutaneous tumour nodules and a few *café au lait* areas. Figure 2—Radiograph of left hip showing a shallow dense acetabulum, a translucent area above it, a well marked os acetabuli, diminution of the joint space, a flat, dense, partly subluxated femoral head, and coxa valga.

On examination there were multiple subcutaneous nodules and a few *café au lait* areas (Fig. 1). A number of mobile, tender, subdermal nodules, one to two centimetres in diameter, were palpable in her left groin and round her left knee. The left femur was five centimetres shorter than the right. She walked with a marked dip to the left, and there was a compensatory lumbar scoliosis. The movements of her hips and knees were full, but the left knee was slightly unstable.

FIG. 3

FIG. 4

Case 1. Figure 3—Antero-posterior radiograph of lower end of left femur, showing several areas of increased translucency. The largest of these is a longitudinally disposed ellipse which has bulged the medial cortex. Its poles are connected by a band of bone of normal density. Lateral to it is a wine-bottle-shaped area. Notice the early osteoarthritic lipping on the medial side. Figure 4—Lateral radiograph of lower end of left femur, showing two streaks of increased density merging into normal bone proximally and distally.

The blood count, serum calcium, serum phosphorus and serum alkaline phosphatase were normal. The radiographic changes (Figs. 2 to 4) were confined to the left hip and femur, and were therefore monomelic. The hip showed malformation and the femur contained several areas of increased translucency. Early osteoarthritic changes were present in the left knee.

After a two months' course of short-wave diathermy, quadriceps exercises and a single injection of ten millilitres of 2 per cent procaine into the nodules in the left groin, the patient had negligible symptoms.

Case 2—A forty-year-old man had pain round his left ear and cheek; this was associated with a lower motor neurone lesion of the left facial nerve due to geniculate herpes, from which he recovered completely within five weeks. He also suffered from neurofibromatosis, without disability. No other member of his family was affected.

On examination the skin changes were like those in the first case. His left tibia was 4·5 centimetres longer than his right, but his femora were the same length. There was marked forward bowing of his left tibia (Fig. 5). The front of its lower half was covered with a firm, lobulated swelling tightly stretching the skin without adhering to it. The swelling was 18 centimetres long, 9 centimetres wide and up to 5 centimetres

FIG. 5

Case 2—Photograph demonstrating the forward bowing of the left tibia and the soft-tissue swelling covering it.

FIG. 6

FIG. 7

Case 2. Figure 6—Lateral radiograph of the left tibia, showing cortical sclerosis and thickening and anterior roughening. Figure 7—Lateral radiograph of the left ankle, showing early osteoarthritic lipping at the ankle and talo-navicular joints and roughening of the upper border of the talus.

deep. Its margins blended with the tibia. His left foot and ankle were clinically normal. He dipped to the right when he walked, and he had a compensatory lumbar scoliosis.

The radiographic changes, again monomelic, were confined to the left tibia and talus. The tibia showed forward bowing, cortical sclerosis and thickening (especially anteriorly) and a radiolucent area in the medullary cavity just below its midpoint (Fig. 6). Early osteoarthritic lipping was present in the ankle and talonavicular joints. The dorsal surface of the talus appeared roughened (Fig. 7).

DISCUSSION

About one-third of the reported cases of skeletal neurofibromatosis have joint changes, which fall into three main groups: 1) dysplasias; 2) sclerosis of the articulating bone end; and 3) secondary osteoarthritis.

Dysplasias have been recorded thus:

At the hip—Coxa valga (Brooks and Lehman 1924), coxa valga associated with a shallow acetabulum as in Figure 2, coxa vara (Uhlmann and Grossman 1940), congenital dislocation (Holt and Wright 1948).

At the foot and ankle—Calcaneus deformity associated with distortion, underdevelopment and displacement of all the tarsal bones (Moore 1941), congenital rocker foot (same author),

congenital flat foot due to misshapen tarsal bones (same author), lateral displacement of talus and calcaneus (Brooks and Lehman 1924).

In the hand—Hypertrophy of the bones of the first and second digital rays associated with expanded bone ends (McCarroll 1950).

Sclerosis of the articulating bone ends, though often accompanying the dysplasias, has been observed as the chief abnormality at the following joints:

At the hip—Sclerosis of the acetabulum associated with several radiolucent areas (Friedman 1944).

At the ankle—Sclerosis of lower part of tibia and talus (Holt and Wright 1948).

At the wrist—Sclerosis at distal end of radius, and sclerosis and hypertrophy of the carpal bones (McCarroll 1950).

Secondary osteoarthritis may occur in:

1) *Abnormal joints*, e.g., the coxa valga described by Fairbank (1950).

2) *Normally developed joints subjected to abnormal forces*, e.g., a short femur causing rocking of the knee with every step as in Case 1, or forward bowing of the tibia producing abnormal compression of the ankle as in Case 2.

Incomplete though this list of joint changes may be, it is nevertheless noteworthy that a fairly thorough search of the literature has revealed no record of shoulder or elbow lesions in patients suffering from neurofibromatosis.

SUMMARY

1. Two patients with monomelic bone and joint changes in neurofibromatosis have been described.

2. The joint changes in neurofibromatosis include dysplasias, sclerosis of the articulating bone ends and secondary osteoarthritis.

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SALMONELLA DUBLIN AS A CAUSE OF CHRONIC OSTEOMYELITIS

Report of a Case

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The problem of treatment of chronic osteomyelitis with sinus formation, with or without sequestration, is one of the most difficult that confront the orthopaedic surgeon. The disease is often resistant to all forms of treatment and amputation may be necessary to forestall the development of general effects of chronic suppuration. The case to be described suggests that our views on the causative organism in at least some of these cases are too stereotyped, and a more thorough search may show that the common organisms—staphylococcus aureus, streptococcus, pneumococcus—are not always responsible.

CASE REPORT

A man aged twenty-nine years attended the orthopaedic clinic with discharging sinuses on the front and lateral aspects of his left thigh of fourteen years duration. He gave a history of subacute osteomyelitis at the age of twelve years, which was treated by incision, gauze packing and rest in a Thomas splint. About two years after he had been discharged from hospital a fresh abscess developed below the original site and he was again admitted to hospital. No bacteriological records are available. Two years ago he received a short course of penicillin therapy which lessened the discharge but did not heal the sinuses.

On examination, the left thigh and calf muscles were considerably wasted. The scars of two incisions were present on the thigh, the upper one being healed and the lower one broken down to form a small sinus. Other larger sinuses were present on the front and back of the thigh. The exudate was sero-purulent. The knee joint was ankylosed in full extension.

Radiographic examination showed two large sequestra involving the full diameter of the midshaft of the femur with involucrum formation in which the sequestra were almost completely embedded. Bacteriological examination of the pus showed staphylococcus aureus which was found to be resistant to penicillin but sensitive to streptomycin.

Treatment—Sequestrectomy was undertaken, the sinuses and much diseased tissue were excised and the wound was closed completely. The limb was then immobilised on a Thomas splint and a course of streptomycin begun.

Progress. The wound broke down in several places, forming new sinuses, and the posterior and lateral sinuses had opened up further. Meanwhile a bacteriological report on the examination of the excised material indicated an almost pure growth of a coliform bacillus of the salmonella type, no staphylococcus aureus being found in the specimen. The same organism was isolated from pus from the recent sinuses, but no organism of the salmonella group was discovered in the faeces. The organism was investigated further and identified as Salmonella Dublin. Because of the rarity of this organism as a pathogen, confirmation of identity was sought from the Salmonella Reference Laboratory and was found to be correct. The organism was sensitive to chloromycetin and it was decided to try the effect of this drug. Doses of 0·6 gramme were given every four hours and continued for thirty-six days. The discharge from the sinuses ceased after fourteen days and the sinuses were completely healed when the drug was discontinued. Four months after the operation serum agglutination against Salmonella Dublin " H " was positive at 1 50.

DISCUSSION

The clinical course and bacteriological finding suggest that this was a primary salmonella infection of bone which became secondarily infected by staphylococcus aureus. Other cases of bone infection by salmonella have been described by Krauss (1947), and in a review of the literature Ramon Guerra, Peluffo and Aleppo (1940) found fifteen authentic examples. Vinke and Downing (1947) described a case of infection of the knee joint by an organism of the salmonella group.

My thanks are due to Mr J. H. Penrose for permission to publish this case, and also to Dr R. Ewart Jones and Dr A. J. Beale for their work in bacterial identification.

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DYSPLASIA EPIPHYSIALIS MULTIPLEX IN THREE SISTERS

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Dysplasia epiphysialis multiplex, first described by Fairbank (1947), is characterised by dwarfism, stubby digits and mottling or irregularity in density and outline of the developing epiphyses. The title was introduced in order to bring into one group a number of cases which had previously been recorded under a variety of names. Fairbank's paper was based on a study of twenty patients. The purpose of this communication is to add a further three cases occurring in one generation. This in itself is hardly sufficient reason for publication, but a study of these three sisters enables comparison of the changes found at different ages to be made, and the recording of some unusual and atypical changes.

The condition is said not to be familial, but in Fairbank's series are included two sisters; also quoted are two brothers (cases of Mr J. A. Cholmeley). Other cases occurring in one family were described under different titles but were considered by Fairbank to fall into his group—two sisters (Gardiner-Hill 1937), brother and sister (Barrington-Ward 1912) and a mother and twin boys (Resnick 1943).

The sisters about to be described are from a family of eight children. The father, who is seventy-five years old and five feet three inches high, suffers from chronic arthritis of the rheumatoid type, but there is no radiological evidence of epiphysial dysplasia. The mother is five feet two inches high and also shows no evidence of the condition. The age and height of the present generation of eight children are shown in Table I.

TABLE I

Age and Height of Eight Siblings

(The affected sisters are indicated by heavy type)

	Age	Height
Theresa	4½ years	3 feet 5 inches
Anne	9 years	4 feet 5½ inches
Keith	12 years	5 feet 2 inches
Malcolm	14 years	5 feet 4 inches
Sheila	15 years	4 feet 7½ inches
Robert	16½ years	5 feet 9 inches
Mary	18 years	5 feet 4 inches
Roberta	19 years	5 feet 0 inches

It will be seen that the affected sisters are short compared with the other members of their family. This shortness does not amount to dwarfism, but is sufficiently obvious to enable them to be picked out from their brothers and sisters (Fig. 1). The other obvious features which distinguish them are the short stubby hands and feet (Figs. 2 and 3) and the fact that they all have cleft palates. It is difficult to relate the last finding to their general condition and it is possibly only an interesting coincidence.

FIG. 1

Group of four sisters. Left to right: Anne (nine years) a normal sister for comparison, Sheila (fifteen years), Roberta (nineteen years) and Theresa (four and a half years).

FIG. 2

Figure 2—Sheila's hands showing the characteristic short and stubby fingers. Figure 3—Sheila's legs. The genu valgum was later corrected by a supracondylar osteotomy of the right femur.

FIG. 3

Of the three, Sheila has the severest deformities, and she was brought to hospital because of right genu valgum (Fig. 3). This was satisfactorily corrected by a supracondylar osteotomy of the femur in 1949. When Sheila was under observation the opportunity was taken to investigate the other members of the family.

RADIOGRAPHIC FINDINGS

While epiphysial changes characteristic of the condition are found to a varying degree in all three sisters, there is one striking feature which differs from the description given by Fairbank. He states that " the centres of ossification for the epiphyses may be late in appearing and backward in development and fusion with the shaft may be delayed." In our cases the epiphyses have appeared early and fused early, and a study of the bone detail in the radiographs shows a remarkable degree of maturity for their age. For example, the radiograph of Theresa's hand at four and a half years (Fig. 9) shows a bone age of at least seven years, as indicated by the appearances of the carpal centres. And the radiographs of Sheila's hips at the age of fourteen years (Fig. 5) show fusion of the capital and trochanteric epiphyses, which normally does not occur until the age of eighteen years. This suggests that the relative shortness of these girls may be due to early epiphysial fusion rather than delay in development.

Fairbank, however, emphasised that " the principal change is irregularity in ossification," and these three sisters do show almost all the typical features in this respect.

Hips—In Theresa's case the capital epiphyses are flattened, narrow and irregularly developed (Fig. 4). The neck of the femur is widened and there is coxa vara. The general bony development in advance of her age (four and a half years) can be noted. Sheila's hips (Fig. 5) show a similar abnormality of the femoral head occurring at a later period of growth. Roberta shows the changes to a lesser degree (Fig. 6). The short neck and the beaking of the greater trochanter are seen. *Knees*—Sheila alone has an abnormality of the lower femoral epiphysis causing the knock-knee for which she was treated. The patella on this side is absent. Abnormality in the growth of this bone is also found in the other two sisters. In Figure 7 (Theresa) the precocious and irregular development of the patellar epiphysis can be seen. *Ankles*—The characteristic obliquity of the joint line is not present, but a flattening of the superior articular surface of the talus in the lateral view is seen in all three sisters. *Feet*—The abnormalities of the tarsus and the stunted metatarsals are well shown in Figure 8. *Hands*—The metacarpals are broad and short, especially in Sheila's left hand (Fig. 11). The fourth metacarpal is stunted; it is interesting to note that this finger is not relatively short owing to an increase in length in the proximal phalanx. In Theresa's hand (Fig. 9) the developing epiphyses of the proximal phalanges are narrow and dense. Roberta's hand (Fig. 10) shows less obvious changes than her sisters', but there is a relative overgrowth of the lower end of the ulna. *Elbows*—Sheila's elbows (Fig. 12) show the unusual shape of the lower end of the humerus and head of the radius. In Theresa's elbows at the age of four and a half years the epiphysis of the capitulum, which normally appears at two years, is not yet present. This suggests that the forward subluxation of the radial head is due to lack of development of the capitulum in Sheila's case as well. It may be noted that it is only around the elbow that epiphysial development is delayed; elsewhere it is premature. *Shoulders*—The distinctive abnormality in the shape of the head of the humerus is not present in any of the sisters.

Apart from some flattening of the lumbar vertebral bodies in Sheila, a full radiographic survey of the three sisters showed no abnormalities other than those described.

DISCUSSION

These three sisters show changes in the epiphyses of the long bones which are sufficiently typical to allow a diagnosis of dysplasia epiphysialis multiplex to be made. It is, however, important to note that there is some variation from Fairbank's described pattern in that the

FIG. 4

FIG. 5

FIG. 6

Figure 4—Theresa's left hip. The capital epiphysis is flattened, narrow and irregularly developed. The neck is widened and there is coxa vara. Note the maturity of bony development. Figure 5—Sheila's left hip showing an abnormality like Theresa's, but at a later period of growth. Figure 6—Roberta's left hip which shows some flattening of the femoral head, but the changes are less marked than in the younger sisters.

FIG. 7

FIG. 8

Figure 7—Theresa's left knee, showing the thin and irregularly developed patella. Figure 8—Sheila's right foot, showing the short, stunted metatarsals. The broadness of the first metatarsal and proximal phalanx is striking.

FIG. 9

FIG. 10

Figure 9—Theresa's right hand. The typical shape of the metacarpals is shown. The epiphyses of the proximal phalanges are narrowed and dense. The development of the ossific centres for the carpus is in advance of her age (four and a half years). Figure 10—Roberta's left hand. The stunted metacarpals are typical, but the changes are not so advanced as in the younger sisters. There is overgrowth of the lower end of the ulna.

FIG. 11

FIG. 12

Figure 11—Sheila's left hand. The stunting of the metacarpals is present, the changes being most obvious in the fourth. The middle phalanges are short and broad. Figure 12—Sheila's right elbow, showing the abnormal shape of the capitulum and the consequent subluxation of the radial head.

epiphyses have appeared early and fused early. This is a substantial difference, but since it is impossible to make sharp distinctions in classifications of growth abnormalities it seems correct to include them under this title.

A point of practical importance in consideration of this condition is the suggestion that the changes in the femoral heads may be misinterpreted as pseudocoxalgia. Although there may be radiological similarities at certain stages of the disease, confusion should not arise on clinical grounds. In no case of dysplasia epiphysialis multiplex have there been noted any symptoms referable to these changes. This is confirmed by the three sisters who have never had pain in the hips or limp and who, at their various ages, have full hip movements. It may also be reasonably deduced that in these hips there is a tendency towards improvement without treatment. It is, of course, possible that the sisters may have been affected in varying degrees from the onset, but on the other hand a study of the radiographs suggests that they show serial changes which are most obvious during the period of active growth. In any event, it is submitted that treatment, in the form of relief from weight-bearing, is unnecessary and should not be contemplated because of the radiographic appearances. Moreover, if dysplasia epiphysialis multiplex is diagnosed early in life there should be no need to give the parents a dismal prognosis, for dwarfism is seldom severe, deformity has been troublesome only in a small proportion of the reported cases, and function is invariably good.

My thanks are due to Mr St J. D. Buxton, under whose care the sisters were, for his help and encouragement in the preparation of this paper.

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UNUSUAL FRACTURE-DISLOCATION OF THE SPINE

Report of a Case

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A man aged twenty-four years was injured in a collision between a motor-cycle and a van. On admission to hospital he was shocked, and complained of pain in the back and "pins and needles" in the left leg. There was no disturbance of control of bladder or bowel.

On examination there were tenderness over the lumbar vertebrae, some weakness in the right lower limb, in the muscles of the hip and knee, and more extensive involvement of the left lower limb (Table I). Sensation was normal on the right side, but was impaired at the medial

FIG. 1

FIG. 2

Radiographs showing the fractures through the pedicles of L. 2, 3 and 4, with marked lateral displacement of the corresponding vertebral bodies.

aspect of the left leg. Reflexes were normal in the right lower limb, but on the left the knee jerk was diminished and the ankle jerk absent. *Radiographs* showed a fracture-dislocation of the lumbar spine at the level of L. 2, 3 and 4. The line of fracture was through the pedicles of the three vertebrae and there was marked displacement of the affected vertebrae to the right (Figs. 1 and 2).

Treatment—Skeletal traction through the tibiae failed to effect reduction. A plaster jacket was applied at the end of two weeks.

TABLE I

MUSCLE POWER AT ONSET, AND THREE MONTHS LATER
(Medical Research Council Grading)

	Right lower limb		Left lower limb	
	November 1950	February 1951	November 1950	February 1951
Hip				
Abduction	2	5	0	4
Adduction	3	5	0	2
Flexion	3 -	5	2	4
Medial rotation	4	5	0	1
Lateral rotation	4	5	2	4
Knee				
Flexion	4	5	2	4
Extension	3	5	0	2
Ankle				
Dorsiflexion	5	5	0	2
Plantar-flexion	5	5	4	4
Subluxation	5	5	0	4
Knee jerk	+	+	+ —	+ —
Ankle jerk	+	+	—	—
Plantar	↓	↓	↓	↓

Progress—Within three months the right lower limb recovered completely, and the left showed some recovery in all muscle groups (Table I). The patient was allowed up at three months, and was fitted with a spinal brace at five months. At that time, there was a slight further improvement in muscle power in the left lower limb. There was residual impairment of sensation over the front and sides of the thigh, and along the medial aspect of the leg.

I am grateful to Mr E. S. Brentnall for permission to publish this case.

FLAKE FRACTURE OF THE TALUS PROGRESSING TO OSTEOCHONDRITIS DISSECANS

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Flake fractures of the talus are uncommon injuries, usually associated with marked inversion or eversion of the foot and ankle, coupled with tearing of the medial [internal lateral] or the lateral [fibular collateral] ligament or of both ligaments. Watson-Jones (1943), relating ninety-seven cases of fracture and fracture-dislocation of the talus and navicular bone in the Royal Air Force in two years of war, noted only eight avulsion or flake fractures of the medial or lateral border of the talus. I should like to add one case, which has progressed to osteochondritis dissecans.

CASE REPORT

A man aged thirty-nine years, stepping out of a brake, underestimated the distance to the ground so that his weight fell on to the outer border of his left foot, turning it inwards beneath him. He felt an immediate acute pain in the outer side of his ankle, but was able to walk from the spot with pain and a limp.

On examination, there were marked swelling beneath the lateral malleolus and tenderness over the whole insertion of the lateral ligament, most marked over the attachment of the anterior and middle fasciculi to the talus and calcaneum respectively. There was no tenderness over the shaft of the tibia, nor over the medial malleolus. Flexion and extension at the ankle were normal, but there was limitation of active inversion by pain and of eversion by swelling. By comparison with the opposite foot and ankle passive inversion was increased.

Radiographic examination—A lateral view did not show any bony lesion. In the antero-posterior view there was evidence of an old inversion injury with a bony fragment beneath the tip of the fibula, and a recent flake fracture of the supero-lateral border of the talus (Fig. 1). There was also a well marked soft tissue swelling over the lateral malleolus.

FIG. 1

Radiograph taken soon after the injury, showing flake fracture of the talus.

Treatment—The ankle was immobilised in a short non-weight-bearing plaster for three weeks, and a below-knee walking plaster for a further three weeks.

Progress—Eight months after the injury, the patient walked without limp or pain, though there was some thickening of the joint and slight tenderness on palpation immediately in front of the lateral malleolus. Further radiographic examination showed abnormal bone formation on the medial aspect of the medial malleolus, doubtless caused by avulsion of the medial ligament from the malleolus at the time of the injury. The flake fracture of the talus had not united, and with the consequent underlying excavation the lesion now resembled osteochondritis dissecans (Figs. 2 and 3).

At this juncture the patient was not anxious for any further treatment. But three months later (eleven months after the injury) he began to complain of pain in the ankle after walking more than one or two miles. Further examination revealed thickening around

FIG. 2

FIG. 3

Eight months after injury. Antero-posterior (Fig. 2) and oblique (Fig. 3) views showing osteochondritis dissecans.

FIG. 4

FIG. 5

Two years after injury. The antero-posterior view (Fig. 4) shows osteochondritis dissecans of the supero-lateral margin of the talus. The oblique view (Fig. 5) suggests that the bone fragment is becoming loose and is beginning to separate.

the joint, and pain on extremes of movement in all directions. Radiographs showed developing osteoarthritis; the lesion of osteochondritis dissecans was still visible, and the fragment appeared to be separating from the talus.

In a further review carried out thirteen months later (two years after the injury) no further clinical deterioration was observed. Radiographs showed osteochondritis dissecans of the supero-lateral angle of the talus, and a small detached bony body in the lateral compartment of the ankle joint. Chronic osteoarthritic changes were present, and the ligamentous injuries already noted were still evident (Figs. 4 and 5). Further treatment is now under consideration.

DISCUSSION

After a moderately severe adduction-inversion injury, the patient suffered a flake fracture of the supero-lateral angle of the talus. Presumably the fragment was buckled off the lateral border of the talus by the contiguous articular surface of the lateral malleolus.

The significant feature of this case is the progression of what was originally a simple flake fracture to a definite osteochondritis dissecans. This direct relationship between injury and the onset of the lesion has already been noted by Vaughan and Stapleton (1947).

The site of separation in this patient was not the most common for osteochondritis dissecans of the talus. Ray and Coughlin (1947) reported fourteen examples of osteochondritis dissecans of the talus, eleven on the supero-medial border, and only three on the supero-lateral. In none of their cases did they record radiographic evidence of a flake fracture of the talus, nor has a study of the literature revealed a similar case.

SUMMARY

1. A case of flake fracture of the talus progressing to osteochondritis dissecans is reported.
2. The relationship between direct injury and the onset of the lesion is noted.
3. The sequence of events was observed radiographically and clinically for two years from the date of the original injury.

My thanks are due to the Director-General of Medical Services of the Ministry of Pensions for permission to publish this case.

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MALIGNANT OSTEOCLASTOMA

Report of a Case

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Ambroise Paré in the sixteenth century probably recognised the condition of osteoclastoma, but the first adequate description was given by Sir Astley Cooper and Benjamin Travers in 1818. Lebert, in 1845, described the characteristic giant cells, but for nearly a century their nature and origin was disputed. Stewart in 1922 stated that they were osteoclasts, a view which has been accepted by most British pathologists, but in the United States this neoplasm is still known by the non-committal name of giant-cell tumour. The two great contributions to our knowledge of this disease were those of Sir James Paget in 1853 and Nélaton in 1860, both of whom considered it essentially benign, as Cooper and Travers held before them; but contemporary opinion refused to accept this and the condition continued to be widely regarded as malignant. In 1862 Virchow agreed than an osteoclastoma was usually benign but stated that it might recur or be malignant; this view is generally accepted to-day but seems to have made little impression at the time. The tumour continued to be called a sarcoma and to be considered malignant, and Gross's study of seventy cases (1879) in which he insisted that it was benign made little impression. It was not until Bloodgood's series of papers were published (1910 to 1924) that opinion began to change. His view that the condition was benign became generally accepted; on his suggestion old names such as giant-cell sarcoma and myeloid sarcoma were abandoned and the tumour appeared in the first Registry of Bone Tumours of the American College of Surgeons under the name he suggested, benign giant-cell tumour.

Although an osteoclastoma usually pursues a benign course there is evidence enough that occasionally it can be malignant. A number of cases claimed to be examples of malignant osteoclastoma have now been published, but unfortunately many of them are open to serious criticism. In some cases the nature of the primary growth or of the metastases is uncertain; in others no necropsy was performed and the metastatic deposits were never examined histologically. Some of these cases had a prolonged course, often with repeated attempts at treatment followed by recurrence, and it has been suggested that the malignancy was due to the development of an osteogenic sarcoma and not to any change in the osteoclastoma. In our case the history was short, there was no interference with the primary growth and no recurrence; necropsy was performed and both the primary growth and the metastatic deposits were examined microscopically and found to be characteristic of an osteoclastoma.

CASE REPORT

A Chinese saw-miller, aged thirty-four, was admitted with a large swelling of the right knee which had been present for over a year. It began as a hard, slightly painful, swelling over the medial condyle of the right femur; it was then fusiform, the temperature of the skin was slightly raised and there was egg-shell cracking on pressure. A radiograph showed an osteoclastoma of the lower end of the right femur (Fig. 1). The inguinal glands were not enlarged and there were no signs in the lungs. Disarticulation of right hip was performed. Histological examination confirmed the diagnosis. The wound healed without complications, a tilting-table artificial limb was fitted and the patient was discharged. Twenty-one months

FIG. 1

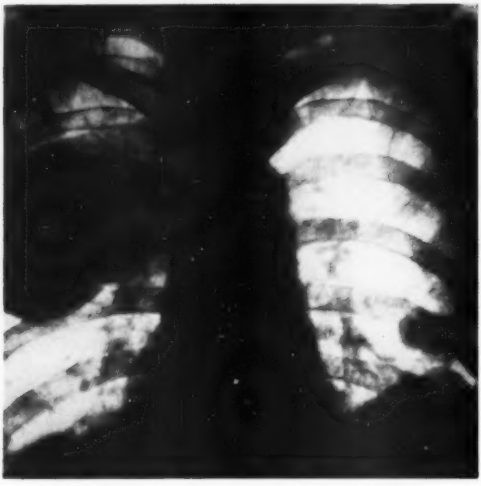

FIG. 2

Figure 1—Radiograph of the osteoclastoma of the right femur. Figure 2—Radiograph of the chest, twenty-one months after the patient's discharge.

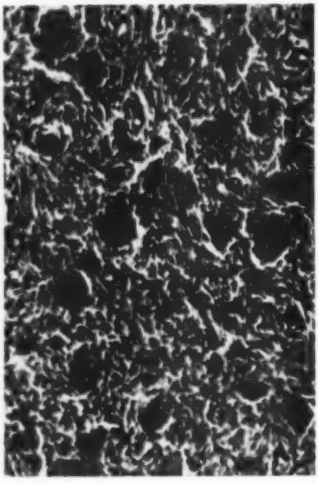

FIG. 3

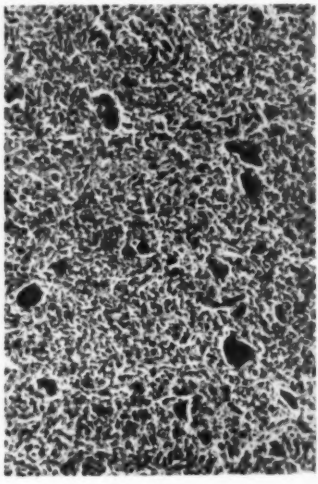

FIG. 4

Figure 3—Giant-cell tissue of the primary tumour (× 106). Figure 4—Giant-cell tissue of a pulmonary metastasis (× 106).

later he was readmitted with a history of having coughed up half a pint of blood; a radiograph of the chest then showed almost complete opacity of the right hemithorax and patchy opacities in the left lung (Fig. 2). He was discharged after further investigation, but returned two months later and died.

Necropsy—The wound was soundly healed and the lymph glands in the right groin were normal. A soft haemorrhagic growth occupied the greater part of the lower lobe of the left lung, and there were three smaller deposits in the left upper lobe. In the right lung there were large haemorrhagic growths in the middle lobe and the upper part of the lower lobe.

Histology. *Primary tumour*—The tumour was composed of giant cells of osteoclast type in a cellular stroma (Fig. 3). The giant cells were numerous (more than thirty in a low-power field) and contained many nuclei, in some cases 100 or more. The nuclei were small, oval and regular, or larger and vesicular, resembling exactly the nuclei of the surrounding stroma cells. There was a prominent nucleolus; mitotic figures were not seen. The stroma was composed of irregular cells with oval or spindle-shaped vesicular nuclei, often irregular in shape, and with great variation in size; in general the nuclei were hyperchromatic, though there was some variation in the quantity of chromatin. A few mitotic figures were present. The growth was vascular, and there were numerous haemorrhages. In some areas there was formation of a delicate fibrous tissue, but the stroma cells in general did not form any collagen and the fibrous tissue appeared to be a reaction to haemorrhage or oedema. In other areas there was active absorption of spicules of old bone, and in one area there was formation of new osteoid tissue. Some fat cells were present and there were occasional groups of lipoid-laden foam cells. The general appearance was that of an osteoclastoma; only the plump vesicular nuclei of the stromal cells, rather irregular in shape and varying in size and the degree of hyperchromatism, suggested that the neoplasm was not as benign as an osteoclastoma usually is.

Metastatic deposits—The deposits in the lungs showed gross destruction of tissue with necrosis and haemorrhage. One small deposit immediately beneath the pleura was not so damaged and showed a similar appearance to the primary growth (Fig. 4). There were many large giant cells of the osteoclastic type, as in the primary growth. The nuclei of these cells were oval and vesicular and contained a prominent nucleolus; they were identical in appearance with the nuclei of the surrounding stroma cells. Small giant cells with six to twelve nuclei were more frequent than in the primary growth and appeared to be formed by fusion of the stroma cells. The cytoplasm and nuclei of these cells were identical with those of the larger giant cells. There were no giant cells of malignant type. The stroma cells had oval or spindle-shaped vesicular nuclei, generally without a prominent nucleolus. They varied greatly in size and shape, but were smaller than in the primary growth; all the nuclei were markedly hyperchromatic. The proportion of spindle cells was higher, though in any field cells with round or oval nuclei were also present. A few mitotic figures were found in the stroma cells. There was a fine delicate stroma, but little formation of collagen and no formation of bone or osteoid tissue.

DISCUSSION

The histological appearance of this neoplasm was not precisely that of a benign osteoclastoma, but there was sufficient resemblance to enable it to be recognised as an osteoclastoma. The history was comparatively short and it therefore seems more probable that the growth was malignant from the first than that it was originally a benign tumour which later became malignant. This is supported by the absence of all the usually accepted predisposing causes— there were no repeated attempts at treatment, no recurrences, no radiation therapy and no pathological fracture. Death occurred from pulmonary metastases. These have been found in all the published cases. Frequently the lungs are the only site, but in some cases metastasis has been widespread and deposits have also been found in the skin and subcutaneous tissues, the bones, lymph glands and other tissues, as in the case published by Dyke (1931).

Perhaps much of the controversy to-day on the existence of a malignant osteoclastoma is really a dispute on nomenclature. A malignant osteoclastoma can be called an osteogenic sarcoma; a sarcoma is any malignant tumour derived from the connective tissues, and an osteogenic sarcoma is a malignant tumour derived from the specialised connective tissue which we call bone. But such a name gives less information than malignant osteoclastoma, and obscures the fact that an osteoclastoma may occasionally become malignant. If it is desired to use the term osteogenic sarcoma for malignant osteoclastoma some qualification is necessary. Stewart in 1922 suggested the name osteoclast sarcoma; it seems a suitable one, but malignant osteoclastoma is equally appropriate and has been in use for a number of years while Stewart's suggested name seems never to have received any general acceptance.

Willis (1948) has stressed that benign and malignant are merely convenient terms to express the probable behaviour of a neoplasm; they do not represent a fundamental biological characteristic. It is therefore surely wrong to suggest that an osteogenic sarcoma may arise in an osteoclastoma as a completely new and different neoplasm. If an osteoclastoma is malignant or becomes so, it remains the same neoplasm and may still show sufficient differentiation towards the formation of osteoclasts to determine its type. The suggestion that an osteogenic sarcoma arises as a second and unconnected neoplasm in the neighbouring non-neoplastic tissue lacks proof, and the published cases do not suggest that it has ever occurred.

SUMMARY

A case is described of malignant osteoclastoma of the lower end of the femur in which death occurred from pulmonary metastases. The history was short and there was no interference with the primary growth, treatment being confined to disarticulation at the hip. Reasons are given for considering this neoplasm a malignant osteoclastoma rather than an osteogenic sarcoma, and the question of nomenclature is discussed.

We have to thank Dr D. R. McPherson, Radiologist, Federation of Malaya, for his report and radiographs; and the Directors of Medical Services and the Institute for Medical Research for permission to publish this case.

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TISSUE DIFFERENTIATION IN MALIGNANT SYNOVIAL TUMOURS

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"... the variety that is manifested in Human Nature and in human life and institutions is a superficial phenomenon which masks, without impairing, an underlying unity." A. J. Toynbee, *The Study of History*, Part III, C iii.

Tumours composed of different kinds of tissue show different characteristics in rate of growth, infiltration of surrounding structures, frequency of complications such as necrosis and haemorrhage, and development of metastases. Thus the prognosis, either natural or following treatment, of various neoplasms, differs. This applies obviously to tumours of different organs or structures, but is true also of tumours of related tissues. It is desirable, therefore, that we should clearly differentiate tumours which, though different, are apparently similar in kind or origin.

The recognition of the nature and, by inference, the origin of a tumour depends in ideal circumstances on a number of observations—the site of the primary growth, its relation to surrounding tissues, the gross features of the tumour mass and its histological characters. The last two go hand in hand but histological features provide the more important criteria.

The differences in normal tissues (for example, in a limb) are due to differences in the direction and degree of differentiation of their original cells, which may have given rise to fibrous tissue, bone, cartilage or synovial tissue amongst others. In the same way tumour cells may differentiate in different directions, and according to which preponderates so the tumour is named. A most important point here is that any conclusion as to the origin of a tumour is merely assumption, and that, though it is commonly stated in the literature that tumours have arisen from synovial membrane, it will be shown that such statements are unjustified and are liable to give rise to confusion.

Synovial tumours are now easily recognised and their principal clinical and pathological features are known. They constitute a group different and distinct, both clinically and pathologically, from other connective tissue tumours. This paper is intended to be a general review of them and also to emphasise one particular topographical sub-group which has been given inadequate attention.

HISTORICAL NOTE

A review of the literature shows a gradual development of understanding of the nature of these tumours, particularly in the last twenty years. Appreciation of some features was slow, not only because this depended on a thorough knowledge of the nature of synovial tissue itself, but also because, as they are not common, opportunities for examining a sufficiently large series seldom come within the ambit of one individual's experience.

Before 1910 many tumours arising in or near synovial membranes had been described, but these were all regarded simply as connective tissue tumours—the malignant examples usually being called spindle-cell sarcomata. Their diagnosis as synovial tumours depended solely on their close relation to synovial structures, usually of joints. In 1910 Lejars and Rubens-Duval reviewed a series of cases and described these as endotheliomata. They observed spaces in the tumours but regarded the cells lining them as being endothelial and, since the lining of the synovial tissue was regarded at that time as endothelium, this designation was appropriate. In 1913 Tourneux made abstracts of ninety-three cases of synovial tumours.

From this time on there were numerous reports of tumours in synovial membrane and most of these were designated endothelioma or some form of angioma, the characteristic histological structure of synovial membrane being overlooked.

In 1927 Smith made a notable contribution. He pointed out that some tumours contained cells arranged differently from those of ordinary connective tissue tumours and emphasised the characters of the former tissue by applying the term "synovioma" to them. A little later the author (King 1931) discussed the nature of tumours of this kind occurring in tendon sheaths, and emphasis was laid on tissue spaces found in them as a special tissue differentiation and the essential similarity of these to normal synovial tissue was shown. The spaces could be lined by only a few or by closely packed cells just as they occur in synovial membrane in different conditions. In the same year Sabrazès and de Grailly (1931) described several tumours and applied the term synovialoma, and Razemon and Bizard (1931) reviewed a mixed group of forty-five benign and twenty-nine malignant tumours.

In 1938 Berger, in a valuable contribution, gave a classification of these tumours and emphasised both the presence of synovial spaces and areas of mucoid change. These were regarded as two kinds of differentiation of this synovial tissue. In 1942 Fisher reviewed a series of forty-two cases. He emphasised the presence of a definite lining of cells in the spaces of the tumours and applied the term sarcomesothelioma to these tumours. The importance of Fisher's paper is that it drew attention to a special type of tumour and furthermore it included members of a topographical group that is more or less ignored elsewhere. These are tumours which histologically are indistinguishable from those occurring actually in joints, bursae or tendon sheaths, but which nevertheless occur in other parts of the limbs.

Groups of cases have been recorded and reviews of the literature have been given by Knox (1936), Coley and Pierson (1937), De Santo *et al.* (1941), Briggs (1942), Lazarus and Marks (1943) and Haagensen and Stout (1944).

An important paper by Bennett (1947) reviewed a series of thirty-two cases of malignant synovial tumours encountered in the American army during the war and since. This material is well presented and magnificently illustrated. There is a clear statement of three main types of tissue found in these tumours. These types indicate various directions of differentiation and include: 1) the presence of spaces in the tumour; 2) "tufts" or groups of oval or polygonal cells; and 3) groups of masses of epithelium-like cells. It is thus clear that the cytological and histological features of these tumours have gradually become better understood.

CLINICAL AND PATHOLOGICAL MATERIAL

Observations were made on twenty-one tumours, most of which were directly related to joints, bursae or tendon sheaths, although a few were quite distinct from them (Table I). These cases have been observed over a period of twenty-five years. Some of them were investigated and treated, though in others only the pathological material was personally studied. Some have been reported previously—a tumour of the prepatellar bursa (King 1939) and a group of seven cases of malignant tendon sheath tumour (King 1941). These are indicated in the Table by the year in brackets after the case number.

All of the examples included in the present series were regarded as being of the malignant type and most of them proved indubitably to be so. Such examples as were clinically or histologically thought to be innocent, though showing similar features, were excluded since they are often not well defined from inflammatory states.

GENERAL FEATURES

As these tumours are not very common an individual is not likely, except over a considerable period, to see many of them. Furthermore, they vary considerably in gross and histological structure, so that examination of any small series may give a wrong idea of the general character of the group. This has been shown in several accounts in the literature.

In addition to the study of the cases tabled here, therefore, the descriptions of the various interested observers have been considered and the conclusions are derived from a scrutiny of material from both sources.

Age incidence—Most of the cases are found in the third decade and the average age in most groups is about thirty-five years. Differences in individual series are exemplified by the group, described by Bennett (1947), whose cases were largely derived from service personnel; the average age was twenty-eight years. In the description of a special histological group Fisher (1942) stated that cases did not occur under puberty; but in a general group cases have been described in children—for example, a child of seven years (Smith 1927) and a baby of nine months (Coley and Pierson 1937) amongst others. In the present series the age incidence was a little higher than that seen elsewhere (average thirty-seven and a half years) but the majority of the cases (thirteen of the twenty-one) occurred in the third and fourth decades. The average age incidence is lower than that of fascial and related sarcomata.

Sex incidence—In most groups studied there has been a predominance of male patients; thus Coley and Pierson (1937) give the proportion male to female as 9 : 4; Berger (1938) gives 13 : 8; de Santo *et al.* (1941) give 11 : 5; Haagensen and Stout (1944) give 62 : 41 and Lazarus and Marks (1943) give 42 : 33. The clear influence of extraneous factors in determining the relative numbers in any group is well shown in the overwhelming predominance of males in the cases described by Bennett (1947); among thirty-two cases, mostly from the services, there was only one female. In the present series there were twelve males and nine females. In the cases described by Briggs (1942) and reviewed by Fisher (1942) the sexes were affected equally. The conclusion is that any sex difference is not, in our present state of knowledge, of practical significance.

Relation to trauma—An injury to the area in which the tumour develops is described by a certain number of patients. It was concluded by de Santo *et al.* (1941) that such injury initiated symptoms rather than gave rise to the tumour, and Briggs (1942) remarked that the injury described was mild. In the present series there was a clear-cut history of injury in four cases and in three others a minor injury or sprain was regarded as having attracted attention to the condition. In all such cases it is important to appreciate that too much attention must not be given to such uncertain evidence as the memory of a patient who is often shown, in other particulars, to be untrustworthy in the memory of details. In general, it may be said that mechanical injury does not play any significant part in the development of these tumours.

Signs and symptoms—In the majority of cases the patient presents himself for examination and treatment because of the presence of a nodule or tumour which in most cases has been present for only a short time. Occasionally it may have been there for a considerable period; in cases in the present series a swelling was present for three years, four years and ten years respectively. It seems obvious, particularly in such a case as this last, that either the tumour changed its character and began to grow rapidly, or a malignant tumour developed in what had been an innocent tumour or ganglion.

Often the tumours are painless but occasionally, especially in those in close relation to joints, some discomfort is observed by the patient. Less commonly pain may be present and occasionally the mass may be tender.

Location of tumours—Tumours have been found rather more frequently in the lower than in the upper limb. This was remarked particularly by Briggs (1942) though Fisher (1937) collected nearly as many cases in the upper limb. The series recorded by Bennett (1947) shows a considerable predominance in the lower limb and in the present series fifteen of the twenty-one cases were found in the lower limb.

Tumours are usually found in close relation to joints, bursae or tendon sheaths, but though some of them are intimately incorporated in these structures it is important to appreciate that tumours may develop apart from the actual synovial membrane. This has been mentioned

specially by Haagensen and Stout (1944) who indeed emphasised that the tumour does not usually arise in the actual synovial membrane. They referred especially to tumours which are actually in close propinquity to joints or synovial structures but this is important in those tumours which lie at a considerable distance from a joint or bursa. A tumour, for example, lying in a limb half way between joints, say, in the middle of the thigh, cannot be related to any normal pre-existing synovial structure. This distribution has a very important bearing on the proper understanding of the pathogenesis and nature of these tumours and will be discussed again.

MACROSCOPIC APPEARANCES

The tumours vary considerably in size, which depends on the rate of growth and the time between the inception and observation of the tumour. Tumours as large as 20 centimetres in diameter have been described but it is more usual for the patient to seek attention earlier; the tumours are frequently seen when about 4 or 5 centimetres in diameter.

The tumours may be either circumscribed or diffuse. It has been stated that they may be encapsulated, but in the malignant forms this is not so. Tumours grow rapidly and invade and compress surrounding structures and so induce the appearance of a pseudo-capsule which is common to so many, particularly connective tissue, malignant tumours.

In shape the circumscribed tumours are usually irregularly ovoid. They are moulded by surrounding tissues and this gives them a somewhat nodular external appearance. Though many of them consist of a single mass of tissue some are composed of what appears to be an aggregate of nodules. In some cases this seems to be due to separate foci of growth, but in others a similar appearance is produced by disintegration or poor growth of tissue between areas of more active proliferation. In the diffuse tumours there may be an ill-defined thickening of the synovial membrane and this forms a very characteristic type, particularly in tendon sheaths, where it may form a cuff of tissue (Case 10).

Tumours arising near synovial membranes grow away from them rather than into them. It is seldom that a circumscribed tumour projects into a joint and, even if diffuse, tumours cause a thickening of the synovial membrane without encroachment on the cavity; tumours of bursae, however, usually replace the bursa.

Tumours may be firm in consistence and, in areas of fibrosis or calcification, are hard; the more rapidly growing ones, however, are soft and may be friable. Where there is mucoid change or cystic development they are fluctuant. On cross section the tumours show a creamy or greyish homogeneous appearance but in some areas may be more fibrous and white in colour; others show mucoid change which may be of such degree that cystic development is prominent. This may take the form of numerous small cysts or a few larger ones. These large cysts contain a straw-coloured fluid which is sometimes blood-stained. Yellowish areas due to necrosis often occur in the more solid parts of the tumour and haemorrhage is common. Thus on the one hand there may be a tumour of uniform tissue, soft and creamy in colour or fibrous and somewhat whiter, and at the other extreme an irregular cystic mass with a relatively small rim of capsule, the content being mucoid or haemorrhagic. All gradations and variations between these two extreme forms are to be found, so that considerable differences in macroscopic appearances may be observed in this tumour group.

HISTOLOGICAL APPEARANCES

The variety of macroscopic appearances is reflected in the considerable variation in histological structure. The close relations of these various forms are shown not only by the obvious development of each of them from typical synovial tumour tissue but also by their simultaneous occurrence and intermingling in some tumours.

Simple synovial tissue is, of course, a modified connective tissue. A good deal has been made, in some papers, of the morphological distinction between synovial tissue and

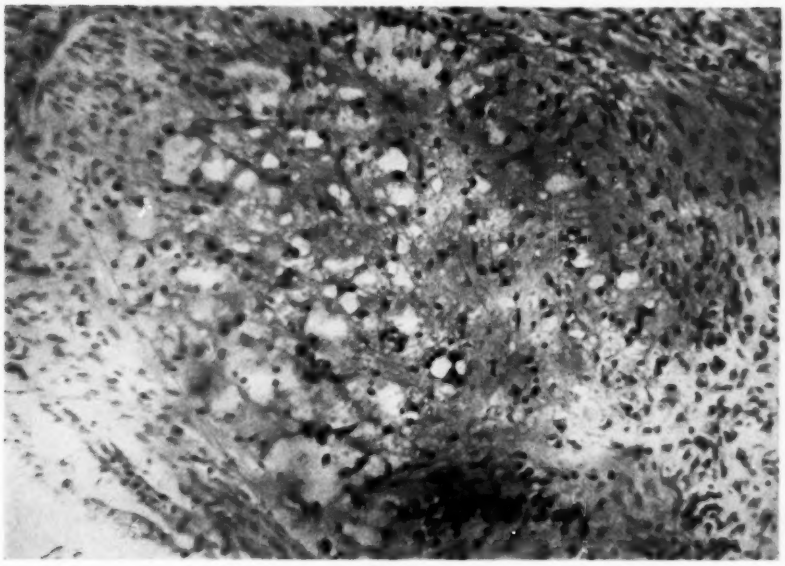

FIG. 1

Photomicrograph of a part of a section taken from Case 10. The material consists principally of mucoid tissue and resembles the early stages of a ganglion. Most of the tumour consisted of material of this kind and despite its benign appearance it was clearly malignant. The small collection of spheroidal cells (which were more numerous in other parts) indicates its activity (× 180).

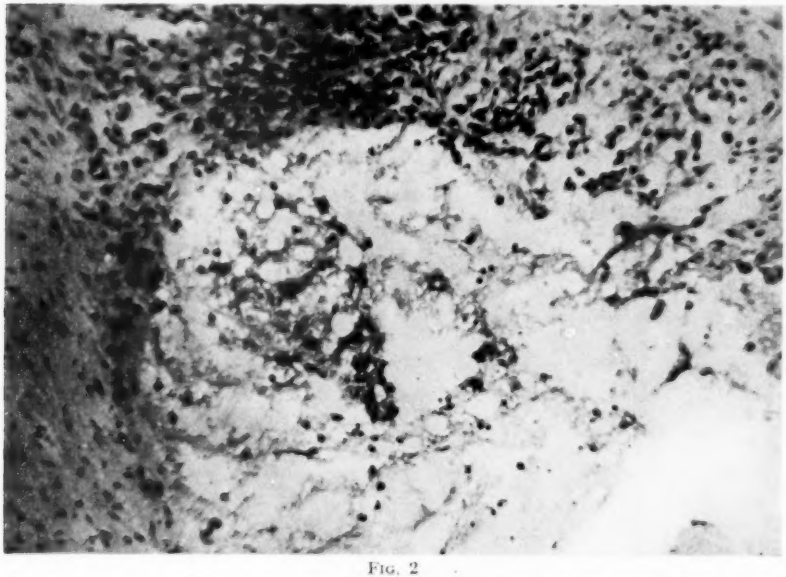

FIG. 2

Photomicrograph of part of a section taken from Case 12. There is considerable mucoid change and the mucinous material is shown by well defined strands of stained material. There are many large spheroidal cells around the area and these merge into the adjacent spindle-cell tissue (× 180).

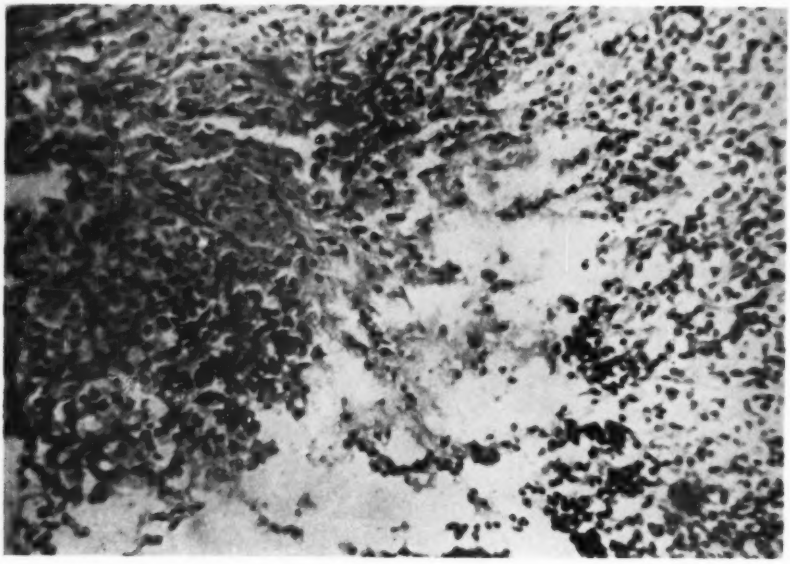

FIG. 3

Photomicrograph of part of a section from Case 5. This shows mucoid change but there is a disproportionately large number of spheroidal cells as compared with the specimens shown in Figs. 1 and 2 (×180).

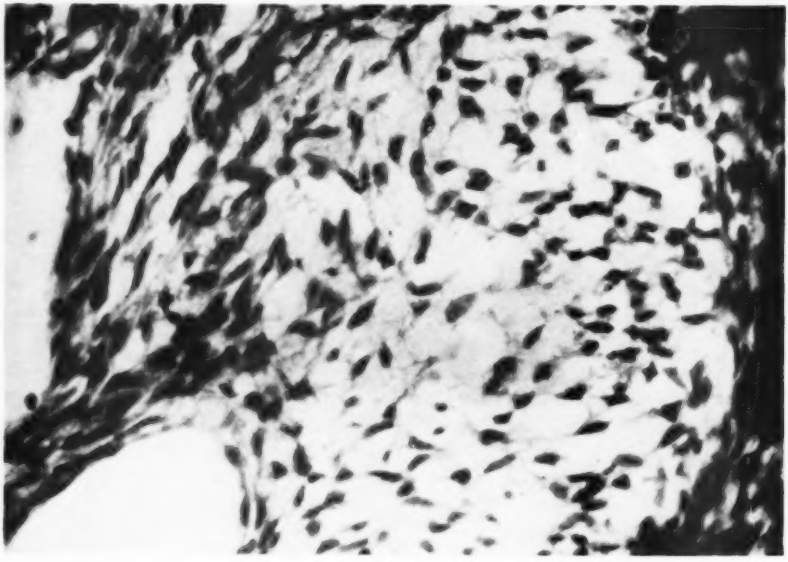

FIG. 4

Photomicrograph of a section of part of a tumour removed from Case 4. The tissue is predominantly spindle-cell in type but there is mucinous material in and between some of the cells. There is also an endothelium-lined space (×400).

spindle-cell connective tissue, as if these two were different kinds. Actually, of course, they indicate two directions of differentiation in these cells. Those near a surface will develop like synovial tissue whereas those away from the surface are of a more indifferent type. It is therefore important, ontogenetically, that we regard the connective tissue—that is, spindle cell tissue—as being the precursor of the other varieties and, indeed, this can be shown to be so.

We have thus a fundamental and pervading spindle-cell tissue and certain other forms differentiated from this. The various forms to be described are:

A. Spindle-cell tissue.
B. Special synovial tissues.
  1. Mucoid tissue.
    (*a*) Cystic form.
    (*b*) Cellular form.
  2. Synovial surface tissue (with typical spaces).
  3. Tissue with endothelium-lined and " gland " spaces.
  4. Spheroidal-cell tissue.
    (*a*) Small cell groups.
    (*b*) " Epithelial " cell strands.
    (*c*) " Epithelial " masses.

A. Spindle-cell tissue—The spindle-cell tissue occurring in these tumours is indistinguishable from that found in a sarcoma occurring in any connective tissue. If there should be no synovial differentiation in any part of the tumour, this cannot be regarded as being synovial but, though a tumour may be composed almost entirely of such spindle-cell tissue, even a small area of differentiation will justify the designation of synovioma.

B. Special synovial tissues—The various forms of differentiated tissue are so diverse that it is easy to understand how a few decades ago the various tumours were regarded as different. Gradations from spindle-cell tissue to these various forms have now been found, so that only the fully formed tissues will be described and, since their character is accepted generally, the accumulated evidence for their inter-relation will not be discussed.

1. *Mucoid tissue*—Mucoid material is found in synovial tumours in at least two distinguishable forms. Doubt has been cast by some writers on the mucoid nature of this material but they have overlooked the observation that synovial mucin is different in its staining (and therefore chemical) character from that, for example, of epithelium. Its gross characters, where such can be determined, the occasionally found positive staining reactions and the absence of positive tests for other protein or fatty material, make its nature certain. It is noteworthy that in such a large series as that described by Bennett (1947) tumours showing this kind of change should have been absent. They were, however, well described by Berger (1938).

(*a*) *Mucoid cystic areas* occur as the most easily recognisable form of mucoid change in these tissues (Figs. 2 and 3). Usually there is a transformation of spindle into polyhedral cells which contain considerable protoplasm in which droplets are to be found. These cells merge into a less cellular area in which dissolution of the tissue has occurred (Figs. 2 and 11). The cells become more swollen and less defined and amongst them there are threads and strands, which material (by its staining character) is typically mucin. Most of the material, however, fails to give staining characters of mucin.

When there is a relatively large amount of this mucinous material the appearances may resemble those of a ganglion (Fig. 1), but in some part there will be cellular areas and, if the condition be a true tumour, it will act as such and, despite its resemblance to a ganglion, will nevertheless recur, invade or metastasise. This occurred in Cases 10 and 15 of the present series.

(*b*) *The cellular type* consists of groups of large spheroidal or irregular cells containing a large amount of clear protoplasm in which there are droplets (Fig. 10). In other places the cells

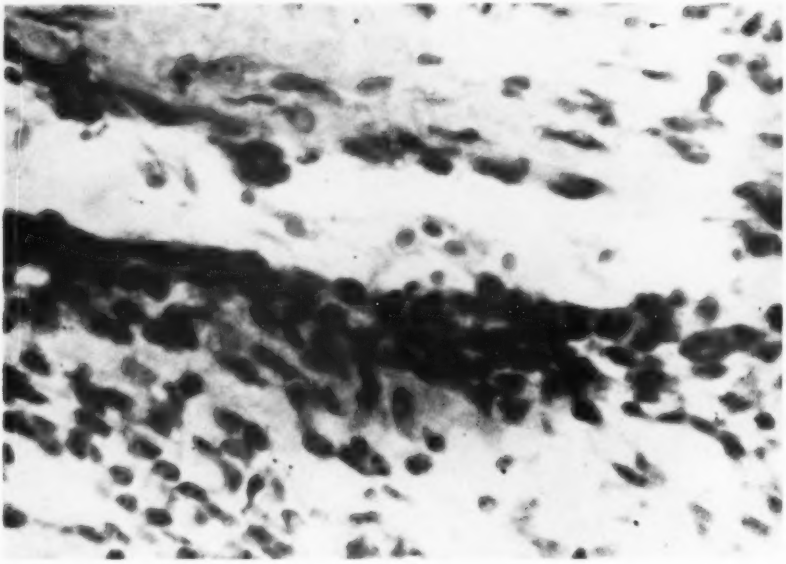

FIG. 5

High power photomicrograph of part of a section from Case 1. There is a well defined cleft in the tissue, one side of which is relatively acellular but the other side shows closely arranged large cells. Since the cells occupying the space are staining differently from the remainder and are separated from each other by more homogeneous and less solid material, the cleft appears to be in the stage of formation (×650).

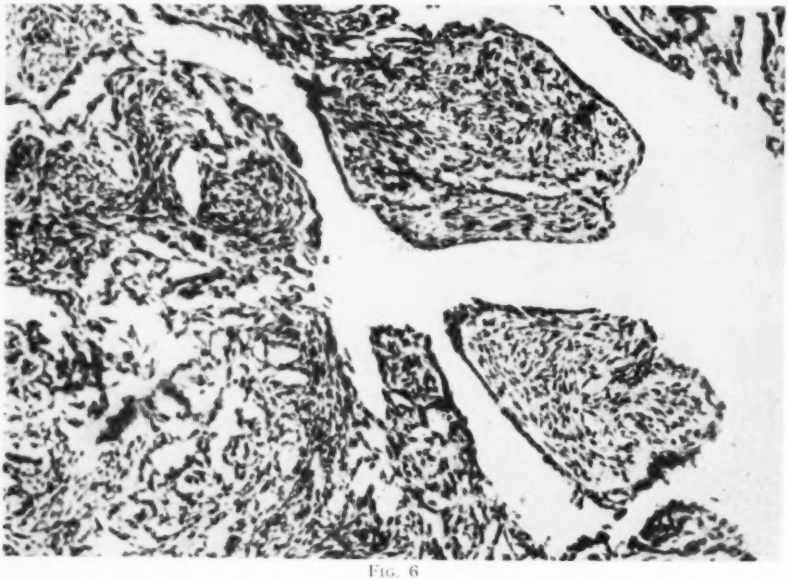

FIG. 6

Photomicrograph from a section of part of the tumour removed from Case 17. The tissue is cellular and there are large branching spaces running through it. In addition there are smaller epithelium-lined "gland" spaces (×140).

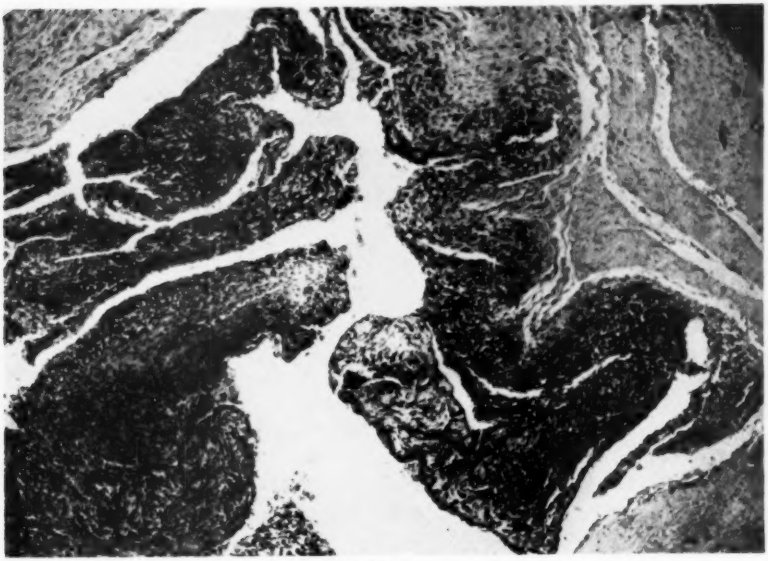

FIG. 7

Photomicrograph of section of part of a tumour removed from Case 19. The tissue is cellular and there are branching spaces through it (×50).

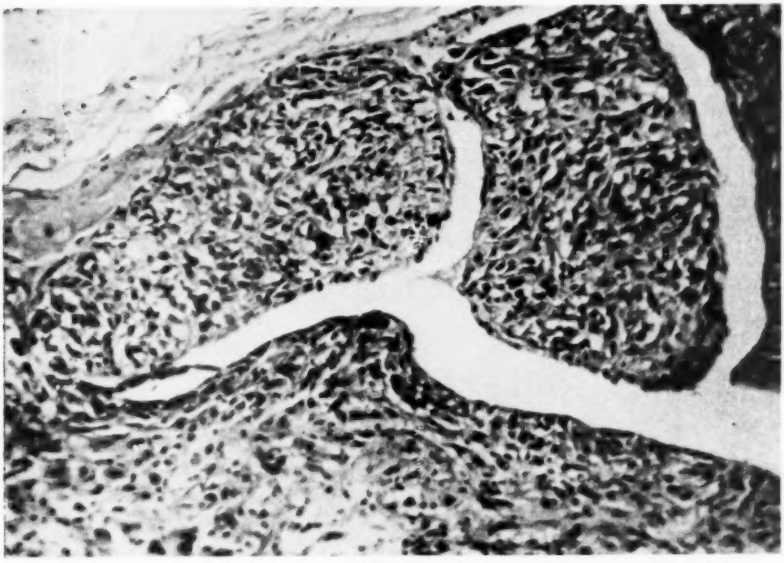

FIG. 8

Photomicrograph of a section of part of a tumour removed from Case 6. The tissue is cellular, composed of large irregular spheroidal and polyhedral cells. There is an irregular space the lining of which closely resembles normal synovial membrane. It is noteworthy that this degree of differentiation should occur in what is clearly an otherwise anaplastic and rapidly growing tumour (×140).

are irregular or stellate and lie in a widely spaced fibrillar stroma in the interstices of which there is non-staining mucoid material (Fig. 4). Case 5 of the present series was almost entirely of this type. This kind of tissue may merge into that described above, in which cystic spaces are found. It might be considered that these changes are merely those of mucoid development in connective tissues and not indicative of synovial differentiation; however, the relation of type (*a*), through ganglion, seems to be clear and, as mentioned, type (*b*) is related to (*a*) in some tumours.

2. *Synovial surface tissue with spaces*—Spaces found in tumours possess a lining which closely resembles synovial tissue; indeed they often show, in miniature, all the characters of synovial cavities; they may be so small as to be found only on histological examination but at other times may be sufficiently large to be obvious as macroscopically demonstrable splits in the substance of the specimen. Though often simple slit-like spaces or rounded cavities, at times they are complicated and branching (Figs. 6, 7 and 8).

The lining is different in different examples. The simplest form resembles an ordinary membrane—that is to say, it is a simple connective tissue surface, some odd cells being on the surface but most lying below it and connective tissue actually lining the surface. It is necessary to distinguish such spaces from those artificially produced. The presence of collagenous tags in artefacts and areas lined by cells in the true spaces make the distinction reasonably easy. Occasionally, early spaces, which indicate their mode of formation, are seen (Fig. 6). In other instances the tissue is more cellular and the cells are plump and relatively large. When numerous, the cells are orientated to the surface, their long axes being at right angles to it so that it closely resembles the membrane of inflamed joints.

Although in some cases tissue cellularity extends from the surface of these spaces out into the surroundings, in others there is a definite distinction, either in the number of cells or in their type, between the two. This gives a further tissue resemblance to synovial membrane. The cells are sometimes more definitely demarcated from their neighbours and resemble either an endothelium or even a columnar epithelium.

3. *Endothelium-lined spaces and "glands"*—For many years, even after adequate descriptions of synovial membrane had been given, considerable confusion occurred because its real nature was not appreciated and it was thought to be lined by a definite endothelium. For this reason the older name for the tumours of synovial membrane was endothelioma. It is important to appreciate now that occasionally the lining of the synovium may undergo change to one with cells indistinguishable from endothelium.

Whether these cells should be called endothelium or epithelium is largely a matter of definition of terms. Since the cells referred to here are spindle in form, have ovoid nuclei, lie along the surface, are derived from connective tissue, and furthermore, since the spaces resemble lymphatics or blood vessels, the term endothelium conforms with general usage (Fig. 13).

There is, however, a still further differentiation. Cells may become cuboidal or even columnar and are much better demarcated from the subjacent tissues than those previously discussed (Fig. 14). Also they are observed, in section, to line relatively small spaces (Figs. 14 and 15). The general form of the cells, particularly their distinction from the adjacent tissue, is one usually regarded as characteristically epithelial (Fig. 15). These occur in the group that Fisher (1942) described and designated sarcomesothelioma. While his arguments in favour of this term may be appreciated, it is doubtful whether, in view of its emphasising only one feature of a larger group of tumours, it has special value.

That these cells, despite their "epithelial" form, are actually connective tissue in origin and are closely related to typical synovial membrane cells is shown, as mentioned previously, by the association in some tumours of the epithelium-lined glands and synovial spaces.

It is sometimes suggested that the use of terms such as carcinosarcoma by some of the older observers is an indication of their lack of knowledge of the subject, but it is clear that,

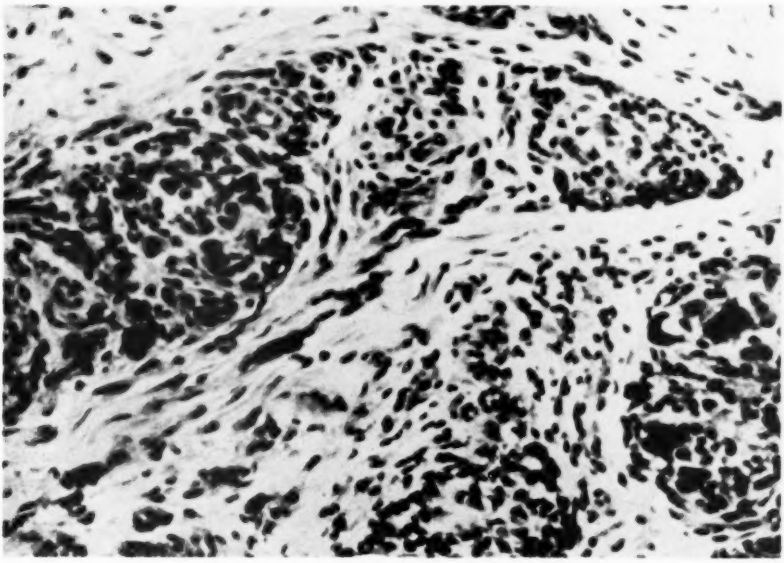

FIG. 9

Photomicrograph of a section of part of a tumour removed from Case 7. In this area there are groups of closely packed spheroidal and polyhedral cells arranged in a more fibrous stroma. Other parts of the tumour showed synovial spaces (×210).

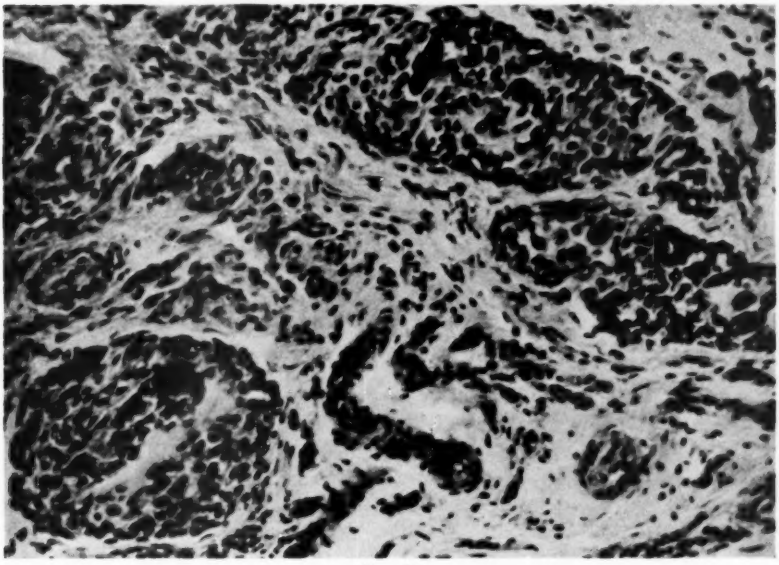

FIG. 10

Photomicrograph of section of part of a tumour removed from Case 2. There are groups of polyhedral and spheroidal cells lying in a fibrous stroma. In general form, this part of the tumour shows a resemblance to carcinoma (×210).

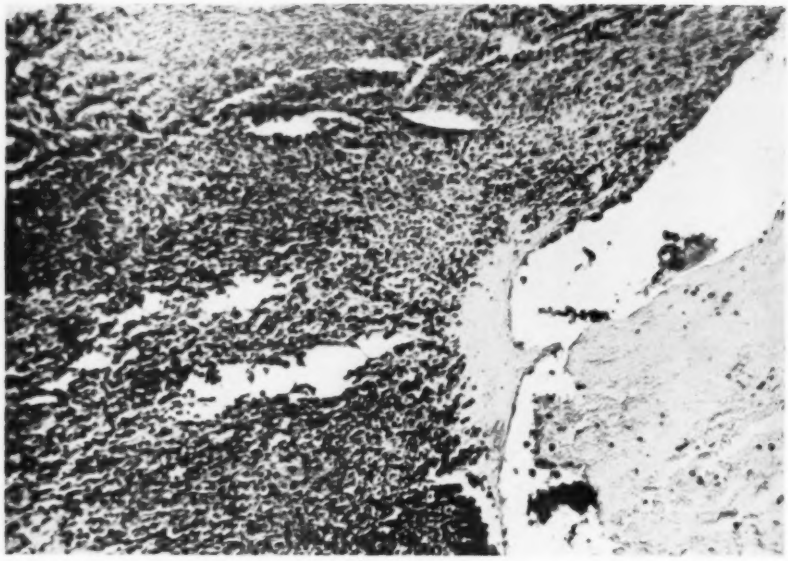

FIG. 11

Photomicrograph of a section of part of a tumour removed from Case 15. The tissue was very cellular and is composed of spheroidal and polyhedral cells, closely packed together, showing a considerable amount of protoplasm. On one side they merge into mucoid tissue which was a striking feature of the tumour. This tumour differs from those shown in Figs. 1, 2 and 3, only by the relative amounts of cellular tissue and mucin ($\times 70$).

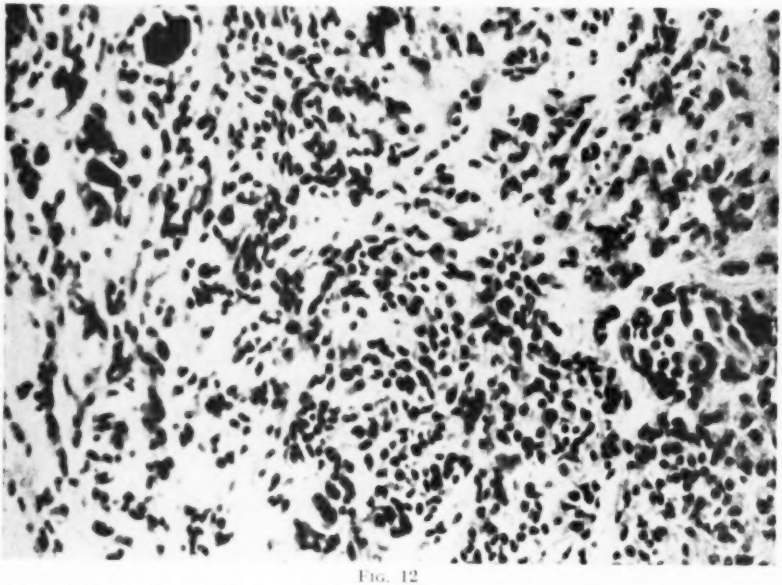

FIG. 12

Photomicrograph of a section taken from Case 18. This is composed of small groups and strands of polyhedral and spheroidal cells lying in the connective tissue stroma. The resemblance of this kind of tumour to a scirrhous carcinoma of the breast may be very strong ($\times 210$).

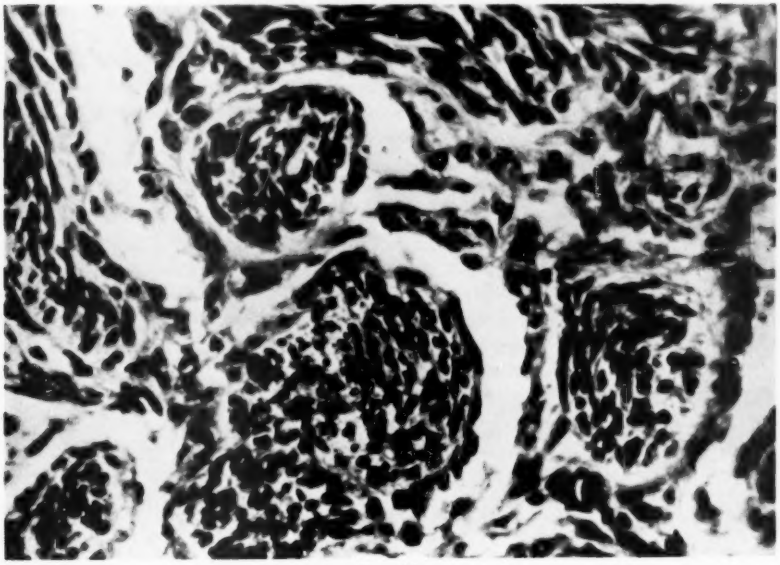

FIG. 13

Photomicrograph of a section of part of a tumour removed from Case 3. The tissue is cellular, being composed principally of spindle cells, the small spaces lined by "endothelium" (×400).

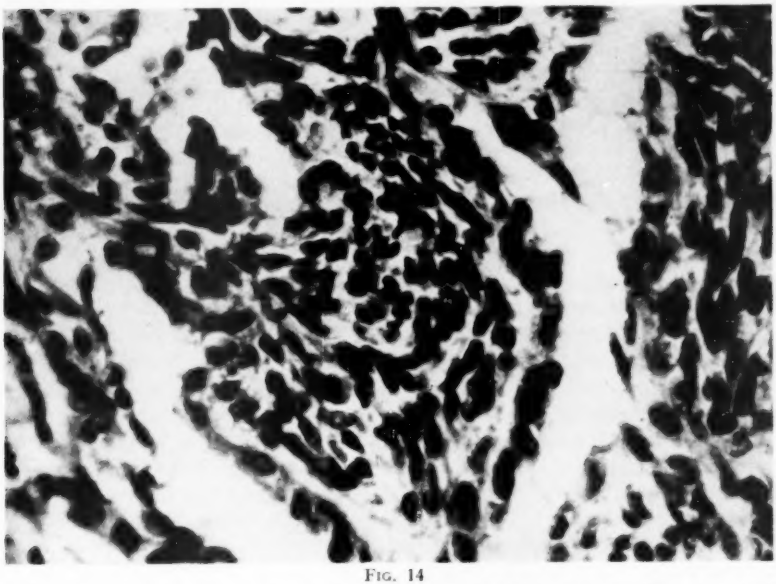

FIG. 14

Photomicrograph of a section of part of a tumour removed from Case 20. There are small circumscribed spaces lined by cuboidal and columnar cells which are well defined from the subjacent tissue and thus can be regarded as constituting an "epithelium" (×650).

on the contrary, within the limits of knowledge of histology at that time it showed a keen observation and appreciation of structure.

4. *Spheroidal epithelium-like cell forms*—Swollen cells of spheroidal, polyhedral or irregularly ovoid form are frequently seen in relation to spaces; in other cases, however, they are found separated from these surfaces. They occur in several forms which may be divided arbitrarily in a number of ways; here they are divided into three main types:

(*a*) *Cell "tufts"* have been described by Bennett (1947). These consist of small aggregations of closely packed ovoid, spheroidal, polyhedral or even spindle cells (Figs. 9 and 10). These may be found producing projections into spaces (Fig. 13) or they may occur as small masses in the surrounding connective tissue. The cell margins are not well defined; the principal feature suggesting epithelium is the absence of a demonstrable intercellular substance. They are important, however, in so far that they merge into the next groups.

(*b*) *Epithelium-like strands*—Spheroidal and polyhedral cells may be scattered through the tissue in strands, often two or three cells thick (Fig. 12). The resemblance of such a tissue, at first

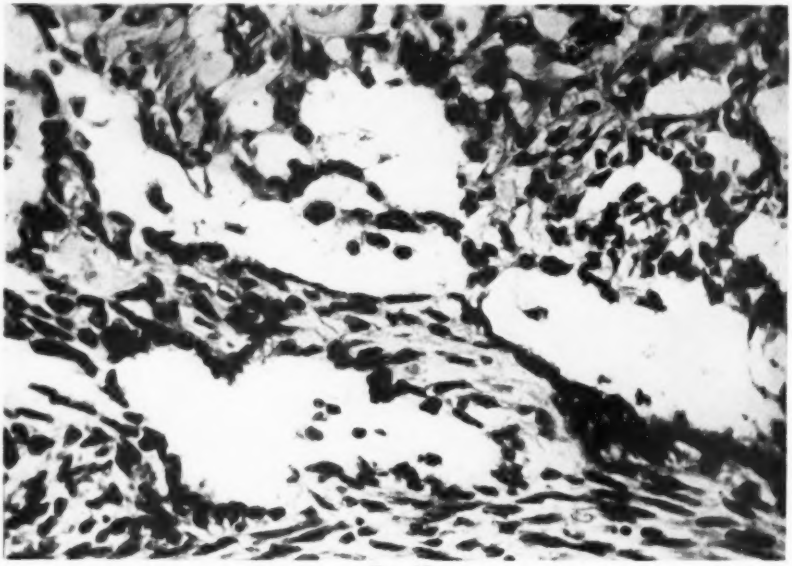

FIG. 15

Photomicrograph of a section of part of a tumour removed from Case 21. Part of the tissue is spindle-celled. There are small spaces lined by an "epithelium" and there is mucoid change in some of the cells (×400).

sight, to a scirrhous carcinoma of the breast may be striking. This similarity was remarked on by Lejars and Rubens-Duval (1910) and a similarity to other carcinomata has been mentioned.

(*c*) *Cell masses* are sometimes large, when they are composed of swollen spheroidal cells which have a well defined cell margin and are clearly demarcated from each other and from the surrounding tissue (Fig. 11). These cells usually show a more voluminous protoplasm in which clear droplets are to be seen and they may merge into large mucoid areas such as were described above.

The various cell types and tissue arrangements described here have been segregated and described separately for reasons of clarity. It happens sometimes that one type predominates and may be almost ubiquitous in a particular tumour but more frequently various forms are to be found in different parts of the same growth.

DISCUSSION

As has been shown, tumours of the synovial type may occur in a number of quite different forms. As a result of the special attention that has been paid to them in the last few years these various types are well recognised as being related to synovial structures.

These tumours are not very common and the various features will only be encountered if a number of cases be examined. It is understandable, therefore, that most authors should have described some features whilst omitting others, but if several papers be examined it will be found that they are complementary and that amongst them all the various features will be discovered.

An important feature of these growths is that a proportion of them may be found away from joints or other recognisable synovial structures. This is emphasised in the present series by the segregation of four cases from the others, these cases not being related obviously to any synovial structure; cases of this type were described also by Fisher (1937). This development of "synovial" tumours in connective tissue away from synovial membranes has an important bearing on the nature and origin of these tumours.

It may be thought to be of academic rather than practical importance how we name tumours; however, the very fact of attempts at classification involving assemblage of similar conditions emphasises its significance. The question is then, how do we classify these tumours? Is it on their nature or on their origin? That there is confusion on this point is shown by reference to the literature. Several authors quite clearly state that it is a matter of the origin of the tumours from synovial tissue. Thus Knox (1936) states that "... a relatively small group [of connective tissue tumours] take their origin from the specialised connective tissue cells which form the synovial linings"; Coley and Pierson (1937) say that "their origin from synovial membrane is now established"; de Santo *et al.* (1941) stated: "Synovial sarcomas originate in synovial tissue and thus are found in joints, periarticular bursae and in tendon sheaths"; Briggs (1942) referred to "tumours which take their origin from the synovial lined structures" and Lazarus and Marks (1943) stated that "synovial sarcomas originate primarily in synovial tissue, and for this reason are found in joints, bursae and tendon sheaths." It will be seen particularly in two of these statements that, because the tumours are named from their assumed origin, they must therefore arise in specific locations.

On the other hand Haagensen and Stout (1944) have "pointed out that, whereas these tumors are composed almost certainly of mesothelial cells, it is very questionable whether or not they are derived from the normal lining cells of joints, tendon sheaths and bursae, because the tumors are usually outside of these structures although often in close proximity to them." It is emphasised here that, though these tumours are often in close proximity to synovial structures, there are the examples which lie at a distance from these and yet give a histological picture which is indistinguishable from those occurring close to the joints. The tumours, however, are not classified on any topographical basis but, irrespective of their position, are grouped together according to their histological structure.

It is obvious then that the opinion that these tumours come from a synovial membrane is pure hypothesis. The only thing of which we can be certain is that they are composed of particular tissues which are microscopically demonstrable. It is important then that we should understand that we classify these tumours according to their histological appearances rather than their presumed histogenesis.

It can be shown readily in these tumours that the various kinds of cells are closely related to each other and may be transformed into each other. Just as in bony tissue bone, cartilage, fibrous and mucoid cells may all arise from one kind of cell, so here we have a similar state of affairs. From the position of some of the tumours it is also clear that these various cells may arise from the ordinary connective tissue in the cells of the limb at a distance from the ordinarily formed synovial membranes.

TABLE I

PREDOMINANT FEATURES IN TWENTY-ONE CASES OF MALIGNANT SYNOVIAL TUMOUR*

Case No.	Sex	Age	Site of lesion	Duration	Macroscopic form	Histological type	Trauma	Treatment	End-result	Time of death
					JOINT TUMOURS					
1	M	32	Knee	4 years, enlarging 1 year	Irregular, ovoid, cystic 6 × 4 centimetres	Spaces, "glands"	—	Amputation	Metastases in lungs	3 months
2	F	26	Knee	18 months, enlarging 1 year	Irregular, ovoid, fibrous 5 × 4 centimetres	Spaces, "epithelium"	—	Amputation	Metastases in lymph nodes	?
3	M	41	Elbow	14 months	Ovoid, fibrous 5 × 3 centimetres	Spaces, "epithelium"	—	Excision	Metastases in lungs	1 year
4	M	29	Knee	6 months	Irregular, ovoid, soft 7 × 3 centimetres	Spaces, mucoid tissue	+	Excision and radium	Recurrence	?
5	F	34	Knee	18 months	Nodular, fibrous 6 × 5 × 3 centimetres	Spaces, mucoid tissue	—	Excision and radium	Recurrence	?
					BURSAL TUMOURS					
6 (1939)	M	35	Prepatellar	3 months	Ovoid, soft 6 × 6 × 4 centimetres	Spaces	—	Excision and radium	Metastases in lungs	2 years
7	F	61	Prepatellar	10 months	Ovoid, firm, whorled 6 × 5 × 3 centimetres	Spaces, "epithelium"	—	Excision	Small recurrence after 3 years	?
8	F	26	Acromial	7 months	Ovoid, fibrous 5 × 4 × 2 centimetres	Spaces, "glands"	—	Excision	Still well after 5 years	
					TENDON SHEATH TUMOURS					
9 (No. 1, 1941)	M	46	Flexor carpi radialis	6 months	Ovoid, fibrous 5 × 4 × 3 centimetres	Spaces	+	Excision	Metastases in lungs	10 months
10 (No. 2, 1941)	M	51	Flexor carpi radialis	12 months	Irregular cuff of tissue 4 × 1 centimetres	"Epithelium," mucin	+	Excision	Local rapid recurrence	?

11 (No. 3, 1941)	F	54	Flexor digitorum pedis	12 months	Ulcerating on surface 4 × 3 centimetres	Spaces	?	Amputation	Metastases in lymph nodes	1½ years
12 (No. 4, 1941)	M	38	Peroneal	2 years	Ovoid, fibrous 3 × 2 × 2 centimetres	Mucin, spaces	?	Amputation	Recurrence, metastases in lymph nodes and lungs	6 months
13 (No. 5, 1941)	F	57	Flexor carpi ulnaris	10 years	Ovoid, fibrous 5 × 4 × 3 centimetres	Spaces, mucin, "epithelium"	?	Excision	?	?
14 (No. 6, 1941)	F	52	Extensor digitorum pedis	3 years	Ovoid, fibrous 5 × 4 × 2 centimetres	Mucin	—	Excision	Recurrence	6 months
15 (No. 7, 1941)	M	34	Flexor carpi radialis	8 months	Ovoid, cystic 2 × 1 centimetres	Mucin, "epithelium"	—	Excision and radium	Cure (12 years)	
16	M	21	Lateral malleolus	6 months	Irregular, ovoid, fibrous 5 × 3 × 2 centimetres	Spaces, "glands"	—	Excision	Cure (14 years)	
17	F	28	Tendo calcaneus	2 months	Ovoid, soft, cystic 10 × 7 × 3 centimetres	Spaces, "epithelium"	—	Excision	Metastases in lungs	1 year
					OTHERS					
18	M	25	Inner thigh (middle)	14 months	Irregular, ovoid, fibrous 8 × 5 centimetres	"Epithelium"	—	Excision	?	?
19	M	28	Inner thigh (upper)	20 months	Irregular, ovoid, cystic 10 × 7 centimetres	Spaces	—	Excision	Metastases in lungs	3 years
20	F	41	Inner thigh (middle)	4 months	Irregular, ovoid, mucoid 10 × 4 × 3 centimetres	"Epithelium" "glands"	+	Amputation	Metastases in lungs	4 months
21	M	31	Groin	5 months	Irregular, ovoid, fibrous 7 × 4 centimetres	"Glands"	—	Excision	Metastases in lungs	8 months

* The various clinical and pathological features of the series of cases which form the basis of this paper. Cases previously recorded are indicated by the number of the case and year of publication in brackets. Sex incidence, male : female, was 12 : 9. Average age incidence was thirty-seven and a half years, the greatest incidence being in the third decade. Duration of symptoms, in general, was only a few months. Most of the tumours were small when first seen. The histological types refer to the predominating features and correspond with the subdivisions given in the text.

This series is described to portray the pathological features of this condition and it is not intended to draw conclusions from the results of treatment, but it is noteworthy that good results were obtained in at least three cases treated by conservative measures. The progress of the tumour and the liability to metastases seem to depend on the nature of the growth rather than on a special method of treatment.

That synovial tissue is more likely to develop in some parts of the body than others in normal circumstances was emphasised by Floderus (1920) when he referred to joints as being developed from an " arthrogenous blastema," but what has become apparent from observations on various conditions, other than tumours, is that this area is not confined to certain localised regions; indeed, experimental embryology shows that the position of tissues in a limb and their relation to other structures is much more important, in determining whether they will give rise to synovial tissues, than any predetermined cellular character.

This of course is a well known phenomenon in ordinary circumstances. False bursae may form in various parts even where normal bursal tissue is not found, for example, on the top of the head. A false joint may be formed in the middle of a bone. These tumours, therefore, which develop at a distance from synovial tissues are thus strictly comparable with these unusual synovial structures.

It may be concluded from this, apart from any other available evidence, that the view that synovial tissue can be formed only from special synovial cells is much too rigid an attitude. Given the appropriate stimulus it seems that connective tissue cells anywhere are capable of this particular kind of differentiation. If we accept this point of view then the occurrence of synovial tumours in areas away from joints and bursae is easily understandable.

In some of the accounts of these tumours they are described as having certain distinct components, commonly a synovial and a fibrous tissue component. This is quite true descriptively and helps to elucidate the complex histological picture, but the suggestion that these are really fundamentally distinct and different is misleading; the fibrous portion of the growth may differentiate into synovial tissue and vice versa.

The term mesothelium, for cells lining some of the spaces in these tumours, has been used by some writers but it is doubtful whether such terms have any real value. These have an embryological significance but their use as applied to structures developing in the adult is rather like applying the terminology applicable to the first four Egyptian dynasties to present day affairs. The tumours show features peculiar to adult normal or inflamed synovial tissue or to other conditions of this tissue (for example, ganglion) and should be named from this rather than from a presumptive and doubtful precursor.

Study of synovial tumours shows that the tumour cells demonstrate many of the potentialities that are present in more " normal " circumstances. In addition they show others that are not often or even ever seen elsewhere, thus demonstrating potentialities which otherwise would not be known. It is now recognised that a neoplasm is not something extraneous, such as a parasite, but is derived from and is part of the body tissues. Thus tumours may give evidence of capacities of cells and provide confirmatory evidence of what has been recognised in other circumstances, that ordinary connective tissue cells in unlikely places are able to differentiate in the direction of synovial tissues.

A principle generally applicable to malignant tumours is that the more differentiated the cells of a growth the less malignant it is. This does not appear to apply, however, to the synovial tumours, because we find a high degree of differentiation of the types described in some of the most malignant tumours. This appears to be due in part to the independence of the degree of cell differentiation and the development of tissue patterns; thus, we may find that in a growth in which there are well developed synovial spaces the tumour cells may nevertheless be anaplastic and atypical. This is well shown in Figure 8. As is found in other tumours the malignancy of the condition is that of the most actively growing and atypical part, so that in assessing the malignancy of any particular tumour it is necessary to consider the characters of any group of rapidly growing cells rather than the general degree of differentiation found throughout the tumour. For the present, therefore, observations on these forms of differentiation are of considerable general pathological importance but in the present state of our knowledge do not help us to differentiate prognostically between different members of the group.

CONCLUSIONS

Synovial tumours constitute a distinct group of connective tissue growths possessing clear-cut clinical features, fairly uniform gross structure and characteristic histological appearances.

The histological appearances vary greatly but they are all due to variants of cell form and variations of tissue pattern which are all derived from changes in simple connective tissue cells. The tissues do not arise from special synovial cells but are due to differentiation of connective tissues which were not of any special type. These tumours, therefore, are connective tissue tumours which show some or other of a series of differentiations in a direction of synovial spaces, fluid (mucin) and special cell groupings. It is thus obvious that they are named from their component tissues and not from an originating tissue.

The appreciation that the classification of these tumours is on the basis of this differentiation, that is, of their structure, rather than of their origin (that is to say, it is histological rather than histogenetic) is fundamental in understanding their distribution and form.

SUMMARY

1. A series of twenty-one cases of synovial tumour is tabled and the histological appearances are discussed.

2. The characteristic histological forms, (*a*) mucin formation, (*b*) synovial spaces, (*c*) endothelial and " gland " spaces and (*d*) epithelium-like tissue, are described.

3. These all arise by differentiation of connective tissue cells which occur throughout the connective tissue part of the limbs.

4. Though more commonly found in regions where synovial membrane is present, they are not confined to such special areas.

5. The tumours are classified on a histological and not a histogenetic basis.

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SPECIMENS FROM THE HUNTERIAN COLLECTION

R. J. Last, London, England

7. The Subtalar Joint (Specimens S 100 1 and S 100 2) *

Two specimens in particular illustrate many points of anatomical and surgical interest in this region.

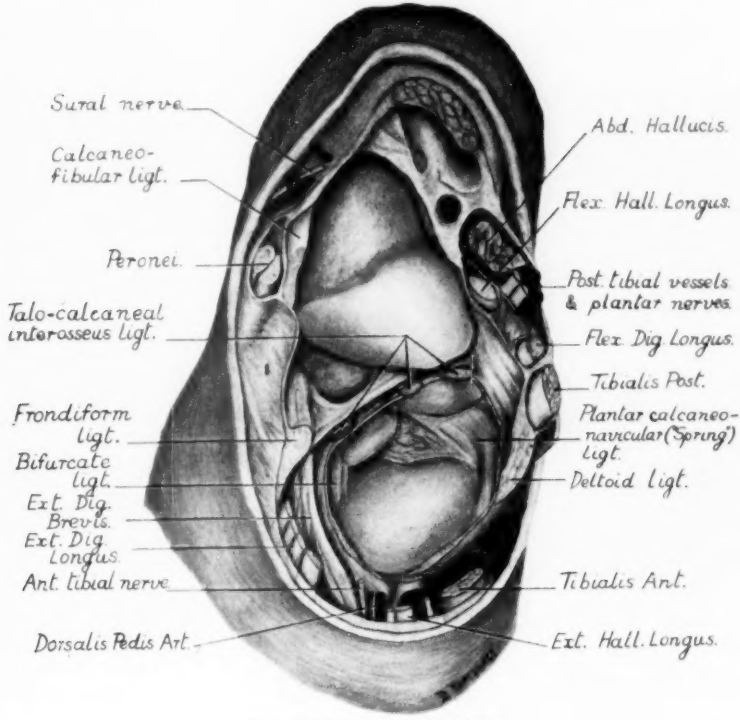
Specimen S 100 1

Specimen S 100 1—In this photograph the right subtalar joint is seen from above. The talus has been freed and lies, inverted, alongside its bed. The structures shown in the photograph may be identified by reference to the Figure. The articulating surfaces of the subtalar joint are exposed. The posterior talo-calcaneal articulation is a simple synovial joint surrounded by a thin capsule; it is the joint by which the body of the talus articulates with the convex upper surface of the calcaneum. Anteriorly the head of the talus articulates with a concave socket consisting of two bones (calcaneum and navicular) and two ligaments (spring and bifurcate). The calcaneum contributes two articulating surfaces to this joint,

* Previous descriptions in this series appeared in Volume 33-B of this Journal, on pages 115 (February 1951), 264 (May 1951), 442 (August 1951) and 626 (November 1951).

the one on the upper surface of the sustentaculum tali, the other on the upper surface of the anterior end of the calcaneum. The wide space between the sustentacular facet and the concavity of the navicular bone is occupied by the fibres of the plantar calcaneo-navicular (spring) ligament, while that between the anterior calcaneal facet and the lateral part of the navicular concavity is similarly occupied by the medial limb of the bifurcate ligament, a strong and constant structure. Between these four areas, the two bony and the two ligamentous, lies a space occupied by a pad of fat (compare the Haversian fat pad in the hip joint). A very convincing account of the movements occurring at these joints has been given by Shephard (1951).

The interosseous talo-calcaneal ligament is displayed in this specimen. It is very strong, and consists of three parts. The strongest lies in the sinus tarsi, connecting calcaneum and



Drawing from SPECIMEN S 100 1

neck of talus, and can be called ligamentum cervicis tali. More medially the tarsal canal is occupied by two strong bands that diverge upwards from the calcaneum to the talus. This part of the interosseous ligament is not well described in most current texts; indeed, its very existence has been denied. It has been examined many times by the writer; it is always present. Its two oblique bands are strongly reminiscent of the cruciate ligaments of the knee joint, except that they do not cross like the letter X but diverge from each other like the letter V. They allow the side to side rocking movements of inversion and eversion, just as the cruciate ligaments allow flexion and extension at the knee. It is probable that these two oblique bands of the interosseous ligament are in no position completely relaxed, and that their tension limits eversion and inversion.

The severed ends of the deltoid and calcaneo-fibular ligaments are well shown. Note the frondiform ligament (of Retzius). It lies superficial to the lateral band of the interosseous talo-calcaneal ligament (ligamentum cervicis tali) and is firmly attached to the calcaneum, whence it is continued upwards over the dorsum of the foot as the inferior extensor retinaculum and downwards over the side of the foot as the inferior peroneal retinaculum (compare Specimen S 135). The extensor digitorum brevis muscle arises not only from the upper surface of the calcaneum but also from the deep surface of the frondiform ligament and by an elongated tendon that lies in the tarsal canal behind the bands of the interosseous ligament. The relations of the tendons and neurovascular bundles in the region of the ankle can be made out readily from this specimen.

Specimen S 100 2—This specimen illustrates very well the structure of the interosseous talo-calcaneal ligament. The head and the posterior half of the body of the talus have been removed. The tarsal canal is thus exposed. The frondiform ligament and the capsules of the

SPECIMEN S 100 2

joints have been removed. The strong ligamentum cervicis tali lies, most laterally, in the sinus tarsi; more medially, in the tarsal canal, the two oblique bands of the interosseous ligament are well seen, the middle one attached to the under surface of the talus, the posterior one attached to the calcaneum in front of its posterior facet; this latter has been divided for removal of the posterior half of the talus. The tendo calcaneus [achillis] is separated from the back of the calcaneum by a bursa; the bony surface is here covered by a very smooth fibrocartilage.

The long plantar ligament shows up. Note the cut tendon of peroneus longus in its true anatomical position, lying not in the groove of the cuboid but bearing on the articular facet on the posterior lip of the groove. The function of these two structures, a tendon and a ligament, in the active and passive support of the *lateral limb* of the longitudinal arch of the foot is self-evident from the study of such a specimen as this.

The base of the fifth metatarsal bone is firmly bound to the cuboid and the fourth metatarsal by strong ligaments which will prevent displacement of the fragment in cases of fracture of the styloid process. In this specimen the tendon of peroneus brevis is cut short at its insertion into the styloid process.

8. Tendons and Retinacula at the Ankle

Specimen S 135—A superficial dissection such as this should be studied in conjunction with such a deep dissection as that of the last specimen. Here the continuation of the frondiform ligament into the inferior extensor retinaculum is illustrated, with the extensor digitorum brevis muscle arising from its deep surface. Note that the base of the fifth metatarsal

SPECIMEN S 135

bone receives, as well as the ligaments referred to above, an expansion from the peroneus tertius muscle which serves further to strengthen the attachment of the fragment after fracture of the styloid process. The tendon of peroneus longus nowhere touches the fibula; at all levels it lies on the tendon of peroneus brevis.

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A METHOD OF HIP ARTHROPLASTY

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The method of mould arthroplasty described by Smith-Petersen in 1939 has been accepted throughout the world. In 1943 Moore and Bohlman published a report of a case demonstrating a metal hip joint, and in 1948 Martin showed a prosthesis consisting of a solid steel head with a screw shank. One year later Robert and Jean Judet demonstrated the use of an acrylic head for arthroplasty of the hip. In 1951 Peterson published a description of another type of solid steel head and shank, the Jaenichen-Collison prosthesis. All these methods have influenced the design of the prosthesis described below.

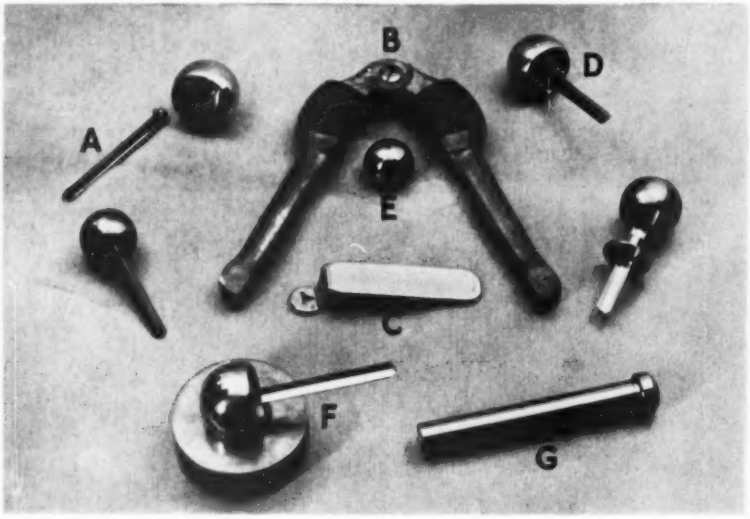

Fig. 1

A shows the cap and nail separately and (below) connected. Note the raised edge on the boss for riveting. B is the special "grip" for holding the head. C is the spanner which fits the trifin nail. D shows the special washer and distance-piece being fitted. E shows the perforation in the cap. F—Prosthesis and stamping plate. G—riveting punch which is threaded over the nail and rivets the nail in place.

THE PROSTHESIS

The prosthesis consists of a hollow steel cap, resembling Smith-Petersen's cup or mould, to the inside of which is fixed a cannulated trifin nail (Fig. 1). The caps are made in four sizes. The nails are smaller than the standard Smith-Petersen nails, the diameter being ten millimetres instead of thirteen. The sharp ends of the nails are tapered and have saw-tooth edges, which prevent splitting of the neck of the femur. The other end of the nail is threaded so that it can be screwed and countersunk into a boss on the inside of the cap, using the special holders and spanner (Fig. 1). The prosthesis is light and strong. The nails are made in different lengths, like the standard Smith-Petersen nails. If an artificial head is desired instead of a cap, a special washer is threaded over the nail and the prosthesis thus changed into a hollow steel head which resembles the Judet acrylic head.

The prostheses of Bohlman, Jaenichen-Collison, Martin and Judet all have solid heads and stems. By their size and shape the stems tend to disturb the nutrition of the neck of the femur. Because of this, it is possible that in the course of years the bone may become damaged or the prosthesis may work loose. These types of prosthesis have an insecure hold on the remnant of the neck of the femur, and it is sometimes difficult to gauge the exact position of the shank in the neck.

The appliance described in this article has several advantages. The trifin shank does little damage to the bone, and since it is cannulated it can be threaded over a guide wire and placed accurately in the centre of the femoral neck. The exact length of stem necessary can be found from the guide wire and a suitable nail chosen. In osteoarthritic hips the head of the bone is not excised but trimmed accurately with a special cutter to take the hollow cap (Fig. 2). This ensures a strong buttress of bone for the appliance. It has already been mentioned that by the addition of a special washer the cap can be used as a hollow steel

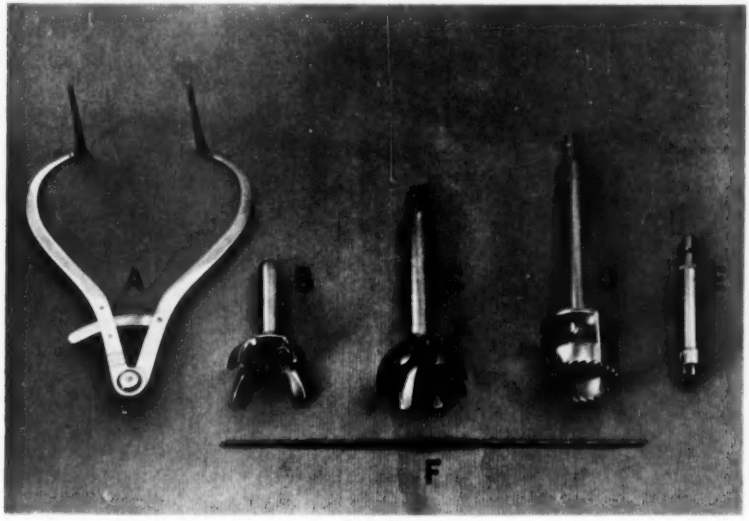

FIG. 2

A—Calipers for measuring the head of the femur. B—Guide through which guide wire (F) is passed into the dislocated head. C—The head reamer. D—A head cutter. There are four sizes to correspond to the caps. E—countersink borer. F—Special guide wire. It is larger than the standard Watson-Jones type.

head (Fig. 1) when it is not possible to retain the femoral head, as in fractures of the neck. (It is possible to fit a trifin nail to a cannulated acrylic head, but this does not seem to have any advantage over the steel one.) The acetabulum is not remodelled unless it is unavoidable, as in bony ankylosis of the hip joint.

TECHNIQUE OF OPERATION

Smith-Petersen's approach is used. Special instruments used are shown in Figure 2. The head of the femur is dislocated in the usual way, and its diameter measured with a caliper. A guide is fitted to the femoral head, and a guide wire is passed downwards through the head and neck of the femur. Radiographs confirm the position of the guide. A head reamer is then fitted over the guide wire and the contour of the head shaped to fit the inside of the cap. A head cutter is then threaded over the guide wire and the circumference

of the head is reduced so that the diameter corresponds accurately to that of the cap to be used. A counter borer is then passed over the guide wire, and a small area cut out to take the buttress on the inside of the femoral cap. A special starter is passed over the guide wire and tapped gently down through the femoral neck. A nail of suitable length is screwed firmly into the femoral cap, and the edge of the boss is tapped over by a riveting punch to prevent the nail becoming loose. The combined nail and head are threaded over the guide wire, and tapped into place with a cannulated punch. The new femoral head is reduced into the acetabulum (Fig. 3) and the incision is closed.

FIG. 3

Radiograph showing the prosthesis in the acetabulum, which has not been deepened or reamed.

Post-operative treatment—The first few cases were treated vigorously. Active movements were begun on the third day and every attempt was made to increase the range of movement as rapidly as possible. Since then a more moderate routine has been instituted. Traction is maintained on the limb, and gentle movements only are allowed after three weeks. "Skating" and assisted active exercises are increased until the sixth week, when the patients are allowed to get up and encouraged to walk in a walking chair. The results of the two methods of post-operative treatment appear to be identical. Since the less forcible procedure is more comfortable for the patient, it has been adopted as the routine treatment.

COMMENT

This operation for arthroplasty of the hip has been used in seventeen cases, the youngest patient being forty-four years of age and the oldest seventy-one. Before operation, all the routine methods of physical treatment were tried and surgery was considered only when there was no improvement. All the patients have been operated upon within the past year, and it is therefore not possible to reach a final judgment on the results. It can be said, however, that most patients are free from pain and have an excellent range of movement.

My thanks are due to Miss Hathway, the superintendent radiographer at the Royal Northern Hospital, who has given much time and thought to the radiographic technique. Messrs Down Brothers made the instruments, and a special word of thanks is due to Mr Maurice Down for his courtesy and patience.

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Sir William Macewen

Sir William Macewen was one of the most versatile of British surgeons. He watched the dawn of antisepsis, grasped its implications and eagerly played a leading part in the romantic expansion of surgery which followed. Many of his widespread contributions were of fundamental importance.

He was born on June 22, 1848, at a house called " Woodend " on Port Bannatyne side of Skeoch Wood, Isle of Bute. He was youngest of the twelve children of John and Janet (née Stevenson) Macewen. His father was a marine trader doing business in sailing ships plying from Rothesay, but family fortune ebbed and flowed like the tide. At one time he was master of the " Breadalbane," a yacht which carried Free Church Ministers to and from the islands of the West Coast of Scotland. The boy, brought up in a seafaring atmosphere, felt the call of the sea all his life, returning to it whenever he could conveniently flee the city. Later in life he bought a small estate on the coast of Bute, engaging in experimental farming and yachting.

John Macewen retired to Glasgow in 1860 and William attended the Collegiate School, Garnett Hill. He was a big, bright and lively boy displaying more prowess in the gymnasium than the classroom; skilful with the single stick. Passing on in 1865 to the University he arrived at a time when the professors in the faculty of medicine were of unusual distinction: there was Allan Thomson in anatomy, Buchanan in physiology, Gairdner in medicine and Lister in surgery. It was the moment when Lister began tentatively to apply carbolic acid to compound fracture wounds, so that Macewen witnessed in Glasgow Royal Infirmary the birth of an antiseptic system which revolutionised surgery. For four years he watched its unfolding, part of which time he was Lister's dresser. Recalling those days Macewen wrote: " Lister's presence in the Scottish universities was of the utmost value. By him teaching was maintained at a high level; he used the universities to stimulate thought, and therein aided them to perform their highest function. It was an inestimable blessing to a university to have such a man in it, and a priceless privilege to the students—to those who could appreciate it—to be allowed to stand silently by and watch the habit of mind and see how the brain worked. He was a man in earnest, and therefore he taught. His teaching was supported by direct appeal to nature. He accumulated data by observation and experiment, from both of which careful deductions were drawn."

Macewen graduated as Bachelor of Medicine and Master of Surgery in 1869 just after Lister had left Glasgow to succeed Syme as Regius Professor of Surgery at Edinburgh. After qualifying he served as house surgeon and house physician before becoming for a short period Superintendent of Glasgow Fever Hospital at Belvedere, an appointment notable for Macewen's introduction of intubation of the larynx through the mouth instead of by tracheotomy or laryngotomy—a procedure that aroused interest at home and abroad whereby he anticipated O'Dwyer's tubes. In 1871 he was appointed a district medical officer, a post which enabled him to gain experience in practical surgery at the parish hospital in Parliamentary Road. Also the same year he became casualty surgeon to the Central Police Division of Glasgow, an office offering him rich experience in emergency surgery and enabling him to contribute many original papers to medical journals, one of which drew attention to a valuable diagnostic sign of alcoholic coma. Macewen had noticed that the pupil of the eye in such a state remained contracted as long as the individual was undisturbed but under mechanical stimulus such as passive movement of a limb, insufficient to arouse from somnolence, the pupil dilated only to contract again when the stimulus ceased. This sign is sometimes referred to as a " Macewen pupil."

He proceeded to the degree of Doctor of Medicine in 1872 and the following year was elected to the important office of dispensary surgeon to the Western Infirmary from which he resigned within a year on appointment to a similar post at the Royal Infirmary. In 1874 he was elected into the Fellowship of the Faculty of Physicians and Surgeons of Glasgow. Macewen was now well set for a surgical career. He started consulting practice at 73 Bath Street, in the centre of Glasgow. In 1876, when he was only twenty-eight years old, he was promoted full surgeon with charge of wards. From that date to 1892 marks the period of Macewen's greatest productivity.

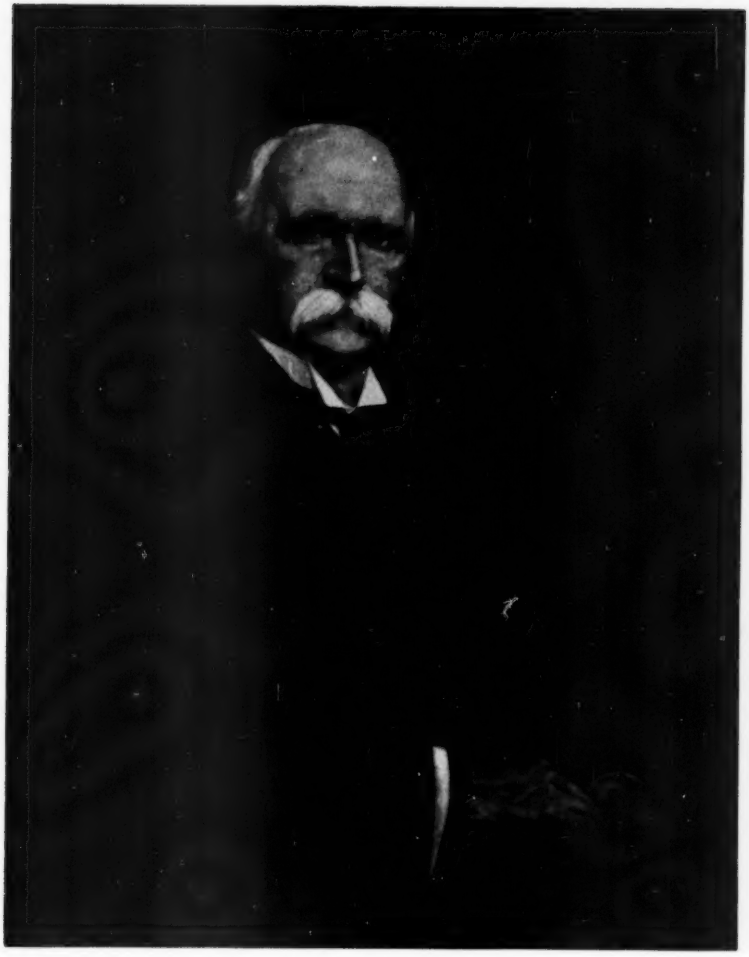
From the beginning Macewen was attracted to the study of diseases and injuries of the skeletal system which in turn compelled him to investigate the physiology and growth of bone, a pursuit he continued until his latter days. During the last century many children in Glasgow, as in other industrial centres, suffered from the evils of overcrowding, lack of fresh air and faulty diet; a social environment that had its reflex in the prevalence of rickets. Macewen became interested in the disease and the pathology of its deformities; he found that the epiphysial cartilages of the long bones were much increased, the diaphyses softened and prone to bend or increase their natural curve under body weight. He shrewdly suspected some error of diet as the causal agent and recommended cod-liver oil, fresh air and sunlight for these young patients, remarking that poor children in the open Highlands escaped the malady. When he contemplated the fixed deformities of severe knock-knee or bow-leg he found that many of them were only capable of correction by operation. The procedure Macewen devised for their cure became the standard operation in surgery; it brought him early world-wide fame.

MACEWEN'S OSTEOTOMY

Subcutaneous osteotomy was introduced by Langenbeck in 1852, when by cutting the neck of the femur he corrected a flexion ankylosis of the hip joint. The first subcutaneous osteotomy performed in England was by Stromeyer Little in 1865 at the Royal Orthopaedic Hospital when, using a mallet and chisel, he corrected a knock-knee deformity. Richard von Volkmann of Halle, the most powerful advocate of Listerian principles in Germany, was the first to perform osteotomy under antiseptic precautions when in 1874 he corrected flexion ankylosis of the knee joint. Macewen, after reading von Volkmann's description of the operation, repeated the procedure in 1875 for a similar condition. Two years later he operated for knock-knee, removing some bone from the inner condyle. On May 10, 1878, he performed linear osteotomy for the first time for correction of genu valgum using a mallet and chisel. The same year in a paper, in the *Lancet*, entitled "Antiseptic Osteotomy," he described this classical operation: "When the limb is extended a point is taken slightly above the level of the upper margin of the patella . . . a more fixed point would be the uppermost part of the external border of the patellar articular surface which can easily be felt beneath the skin, a line drawn about half an inch above this would represent the incision in the bone, the chisel being inserted straight across the femur in that line. When the limb is extended a longitudinal incision is made in front of the tendon of the adductor magnus, the middle of the incision corresponding to the transverse line just spoken of. The length of the incision is slightly greater than the largest chisel to be used." Over two-thirds of the thickness of the femur should be incised before fracture of the bone was attempted. Occasionally a wedge of bone was removed.

Macewen soon found that the chisel bevelled on one side was unsuitable for the straight cutting of bone. He therefore devised an instrument, wedge-shaped at the cutting end, which a skilled craftsman made for him, meticulously tempering it for its purpose; of the instrument he said: "The borders of the blade are marked with half inches, the figures being extremely

We are grateful to the Council of the Royal Faculty of Physicians and Surgeons of Glasgow, for permission to reproduce the portrait of Sir William Macewen.—EDITOR.



William Macewen
1848-1924

light. The figures point out the depth to which the instrument has penetrated and thus serve as a guide. They are finely polished, not for appearance, but because the finer the surface the less opportunity will organic matter have of becoming adherent and afterwards decomposing.

" Now although there is nothing new in the principle on which the instrument is formed, yet its exact counterpart cannot be found out of surgery; and, although clearly of the chisel order, it has sufficient individuality to enable it to take a distinctive name. As the blade in shape resembles somewhat a transverse section of the blade of a pocket knife, and as it cuts much in the same way, it might be described as a knife for cutting bone; or to employ a single word which would at once distinguish it from the ordinary chisel and at the same time be more euphemistic to a patient's ear, it might be called an osteotome."

In making the osteotome an all-metal polished piece Macewen departed from the customary bone-handled or wooden-handled surgical instrument, but it indicates that his mind was already moving towards aseptic surgery. The osteotome has so completely captivated the orthopaedic surgeon as a bone-cutting instrument that the chisel is entirely neglected and its use forgotten. Yet for the controlled shaving and moulding of bone, as in sculpture, the chisel is incomparably the better tool; the osteotome was never intended by its originator for anything but straight fissuring.

Macewen in 1880 published his experiences in a small book, which became a classic, entitled *On Osteotomy: with an enquiry into the aetiology and pathology of knock-knee, bow-leg and other osseous deformities of the lower limbs*. It was translated into several languages. At the International Congress of Medicine held at Copenhagen in 1884 he delivered an address on " Osteotomy for Genu Valgum." He was able to report upon one thousand eight hundred osteotomies without septicaemia or fatal wound complication. These results were received with acclamation and astonishment. They also helped to vindicate antiseptic principles in surgery for which Lister was pleading.

TRANSPLANTATION OF BONE

Some of the best and most abiding work done by Macewen was in connection with the study of the growth and grafting of bone. John Hunter after much patience had wonderfully succeeded in transplanting a human tooth to a cock's comb. Some scanty success was gained by others in the transference of bone from one lower animal to another. But Macewen in 1879 was the first to transplant bone in a human being successfully. It was a great pioneer achievement for at once it opened up a new field in bone surgery. The work was described in a communciation to the Royal Society in 1881 entitled " Observations concerning transplantation of bone. Illustrated by a case of inter-human osseous transplantation, whereby over two-thirds of the shaft of a humerus was restored." This paper is a landmark in surgery; it received the enthusiastic commendation of Professor T. H. Huxley, secretary of the Society, who clearly saw the significance of successful human bone-grafting. In the communication Macewen proceeds as follows:

" William Connell, aged three years, was admitted into the Royal Infirmary, Glasgow, under my care, on the 17th July, 1878, in a much emaciated and exhausted condition arising from suppuration in connexion with necrosis of the right humerus . . . On August 7th, 1878, the part of the necrosed bone exposed at the operation already mentioned was divided by the bone forceps. The upper part of the shaft was then caught by lion forceps rotated in order to loosen its periosteal attachments and then pulled out. The lower portion was similarly dealt with. The two portions removed comprised the whole humeral diaphysis." The wounds ultimately healed leaving the arm flail and useless; the parents requested amputation but Macewen persuaded them to allow him to attempt some improvement of the limb. His narrative continues: " Transplantation of two Wedges of Bone—On November 9th, 1879 (one year and three months after the removal of the necrosed shaft), an incision was made down to the extremity of the upper fragment. This extremity was found to be cartilaginous

for fully a quarter of an inch. This cartilaginous spike like process was removed leaving then a portion of bone which measured 1¾ inch from the tip of the acromion process. From this point a sulcus about 2 inches in length was made in a downward direction between the muscles. The former presence of bone was nowhere indicated and the sole guide as to the correct position into which the transplant was to be placed was an anatomical one. After the sulcus was formed, the haemorrhage was fully arrested and an aseptic sponge was placed in the gap which was then ready to receive the bone.

" Two wedges were then removed from the tibia of a patient six years of age, affected with anterior tibial curves. The last of these osseous wedges consisted of the anterior portion of the tibia, along with its periosteum, the wedges gradually tapering towards the posterior part of the tibiae.

" They were removed, then cut into small fragments with the chisel, and immediately thereafter they were deposited in the sulcus in the boy's arm. They were kept under the spray while they were removed from the tibiae until they were covered by the soft parts of the arm and its antiseptic dressing . . . Result of first Transplant: Two months after, it was thoroughly examined, when a portion of the bone 1 inch in length, and as far as could be measured, nearly three-quarters of an inch in thickness was found firmly attached to the upper fragment of the shaft. In running the finger from the head of the bone towards the graft, the latter could be distinguished by its greater breadth. At this time instead of the former sharp spike the upper fragment ended in an obtuse terminal.

" Placing the arm by the side, the measurement from the tip of the acromion process to the extremity of the bone was now 2¾ inches. There was a distinct gain of 1 inch in length. The arm was shown in this condition to the Glasgow Pathological Society."

A second transplantation of bone was carried out on February 1, and a third on July 9, 1880. "At the beginning of March 1881 the bone was found firmly united, from the head to the condyles and measured 6 inches; while the left humerus measured 6½ inches, that is to say, half an inch longer than the transplanted one. On rotating the condyles the head of the humerus responded to the movement. The patient could lift his arm to his head and otherwise use it."

The late end-result was described by Macewen in 1909 in the *Annals of Surgery*: " It is now thirty years since the humeral shaft was rebuilt and during the greater part of this period the man has depended upon his physical exertions for the earning of his livelihood. He worked as a joiner for many years and now is an engineers' pattern maker. His grafted arm has increased in length, but not proportionate to the increase of the sound one. Measurements: The grafted humerus measures from the tip of the acromion to the tip of the external condyle 10 inches, but following the curve in the bone it is 11 inches long. The sound humerus from the same points measures 14 inches—3 inches longer than the other."

The distal epiphysial cartilage was destroyed by the original disease of osteomyelitis. The increase in length of the diaphysis occurred from the proximal epiphysial cartilage alone. Macewen carried out many successful bone transplantations after this first classic case. In 1903 he succeeded in restoring the transverse ramus of one half of the jaw by transplantation of bone in a girl fifteen years of age who had the horizontal ramus of the lower jaw on one side extirpated for a diseased condition several years previously. He was particularly gratified with the result, for the girl had been restored to her natural good looks from what was a hideous saliva-pouring disfigurement.

THE GROWTH OF BONE

Macewen by his extended researches in the physiology of bone greatly advanced our knowledge of its growth. He proved that bone was a living tissue capable of transplantation; he believed the graft played a vital part during the process of incorporation. In his operative and experimental work he was impressed by the efficacy of multiple small grafts. They provided a greater surface than the massive graft, each forming a centre of ossification which threw

out osteoblasts from its whole periphery. Herein he displays a remarkable insight, for this seems to provide an explanation of the quickened osteogenetic power of small medullary bone-grafts which have found such favour in this last decade.

The growth of a long bone occurred at the diaphysis, for he believed that the cartilaginous growth-disc belonged to the diaphysis and not to the epiphysis. He showed experimentally that the disc was only concerned with the growth of the shaft. He also believed, contrary to Duhamel and Ollier, that the periosteum had no osteogenetic power; it was purely a limiting membrane giving direction to bone-growth but taking no active part in it. He excised bone shafts with the epiphyses in dogs but left the periosteum intact and found that there was no periosteal reproduction of the shafts. In another animal a flap of periosteum was lifted from a radius, detached at its lower end, brought around some muscle fibres and re-attached to the intact periosteum, but the strip produced no bone. Again he removed part of a radial shaft and inserted a glass tube between the remaining segments to exclude the periosteum, and found that osseous tissue invaded the tubes from the severed ends. " The potency of the periosteum as a limiting membrane is seen when, in cases of fracture, it is torn up and stretched across the fractured surface of one of the fragments. It here forms an effective barrier against osseous union, the ossific formation being absolutely limited by the periosteum and fibrous union results." On the other hand, stripping or tearing of periosteum in a fracture allows outpouring of osteoblasts from broken surfaces into the gap between the bones and into the surrounding tissues to form binding osseous deposits. Bone deprived of periosteum will live and grow. Growth and reproduction are an inherent property of the osseous elements themselves. The result of thirty years' clinical and experimental investigation was in 1912 published in a book, *The Growth of Bone: observations in osteogenesis*. This was followed in 1921 by another work, *The Growth and Shedding of the Antlers of the Deer*. The casting of the antlers in early spring followed by the growth of a new pair provided him with the opportunity of closely studying rapid massive osteogenesis in nature.

Macewen was a dresser to Lister at Glasgow Royal Infirmary and saw the effect of the application of antiseptic principles in the treatment of compound fractures, the impressive lowered mortality and the hastened healing of wounds. From that time onwards he became an ardent supporter of Lister, employing in his practice antiseptic lotions and the carbolic spray for several years. But by the middle 'eighties he was already using instruments forged from a single piece of steel and was boiling his gauze. By 1890 he had installed in the hospital a steam sterilizer for dressings after the Schimmelbusch pattern, was boiling his instruments in a fish kettle, wearing a gown and had discarded the spray. He adopted an aseptic technique. Like Lister, he experimented a great deal with catgut and at last succeeded in making a completely reliable and absorbable suture material which he continued to make to the end of his life.

Macewen was about the earliest pioneer of cerebrospinal surgery (neurosurgery). In 1879 he operated upon a patient for the relief of subdural haemorrhage with hemiplegia and the same year removed a brain tumour in a girl fourteen years of age. Both made a good recovery. Another great advance was his recognition that middle-ear disease was a common cause of cerebral abscess. This induced him to design and perfect an operation for mastoid disease. By 1893 he had gathered such a wide experience of this new branch of surgery that he was able to publish an authoritative work entitled *Pyogenic Infective Disease of the Brain and Spinal Cord*. This work was acclaimed all over the world and became a classic. The same year he published another work, *Atlas on Head Sections*, in the fine production of which he spared neither labour nor expense.

Macewen was also one of the first to open the chest and operate upon its contents. He had some experience in dealing with penetrating wounds of the thorax before he attempted a direct attack on the lungs. In 1895 he was asked to see a patient who was desperately ill, emaciated and toxic from extensive active tuberculous disease of the left lung with secondary

pyogenic infection and abscess formation. He performed total lobectomy followed later by thoracoplasty. The man recovered his health completely and afterwards was actively engaged in earning his livelihood for many years.

In 1892 Macewen became Regius Professor of Surgery at Glasgow University, a post that entailed a good deal of teaching and transference of his surgical work to the Western Infirmary. He held the chair until his death. In 1895 he was elected a Fellow of the Royal Society. He was also elected an Honorary Fellow of the Royal Colleges of Surgeons of England and of Ireland; several universities conferred honorary degrees upon him, and he received recognition from leading surgical societies abroad. Soon after the outbreak of war in 1914 he was commissioned as Surgeon-General in Scotland, serving in the Navy with the rank of Surgeon Rear-Admiral. In addition to the onerous duties of this post he threw himself with great energy into organising the Princess Louise Hospital for Limbless Soldiers and Sailors at Erskine, the counterpart in Scotland of Roehampton. In 1922 he was elected President of the British Medical Association on its visit to Glasgow in that year. In 1923 he was elected President of the International Society of Surgeons and later the same year experienced something of a triumphal tour in New Zealand and Australia when he went out to the Australasian Medical Congress at Melbourne. He received the honour of Knighthood in 1902, was made a Companion of the Bath in 1917 and was appointed Surgeon to the King in Scotland in 1909. He died of pneumonia on March 22, 1924. He married in 1873 Mary Watson, daughter of Hugh Allan of Crosshill, Glasgow, and had three sons and three daughters.

Macewen was a man of independent outlook, relying more on his own experience and observation than on the accepted teaching of others. His personality was forceful; he was possessed of immense energy and driving power, prosecuting his work as a scientific surgeon with consuming zeal. His individualism and temperament prevented him from easily co-operating with others in a team; he was entirely happy working alone and work was the breath of his life. He was tall, had a commanding figure and was gifted with a clear, resonant voice, all of which enabled him to hold and often sway any audience he addressed. He was born at a fortunate hour and took full advantage of the opportunities that were presented, turning all his talents to such development of surgery as Lister had made possible. William Macewen's contributions were so varied and of such a quality that he must be regarded as one of the greatest surgeons of all time.

ARTHUR ROCYN JONES.

I am grateful for permission to read the original paper on transplantation of bone in the Proceedings of the Royal Society. I also gratefully acknowledge the great help received by reading the excellently comprehensive biography of Macewen by Dr A. K. Bowman, a work that should be read by every young surgeon.

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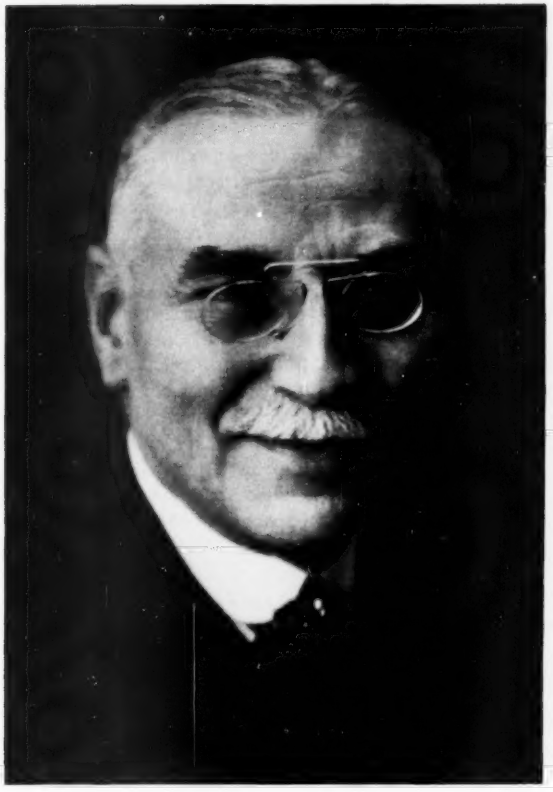

LOUIS TAVERNIER
1877-1951

The death of Louis Tavernier has robbed France of a distinguished son who dedicated his life to the advancement of orthopaedics. He was remarkable alike for his capacity for hard work, for his wide range of interests and the receptiveness of his mind, for the originality of his ideas and his creative power; for his ability, the fruit of sound education and cultivated taste, to express himself in writing with perfect clarity in the many valuable contributions which are a permanent memorial to a man of sound judgment and transparent honesty.

Before the first world war Tavernier carried out pioneer experimental work on certain types of grafting, in particular grafting of the ureters, arteries and the kidney. At that time, however, the technical limitations of suture and the absence of chemotherapy prevented him from transferring the results of this work to Man, except in a few cases of porto-caval

anastomosis which did not turn out well. The outbreak of war gave him the opportunity to turn his attention to the surgery of bone. First in Italy, then in Champagne, he studied every type of wound to determine the conditions under which it was permissible to carry out primary or early secondary suture. In 1915–16, at a time when suppurative arthritis was often fatal in spite of arthrotomy, followed too often by resection of the joint and lastly by amputation, he was one of the first to point out the necessity for selecting the appropriate primary treatment so as to abolish the need for life-saving operations as a last resort. He also studied the pathology of brain abscess with particular reference to its effect on the ventricular system, and the conclusions he reached still hold good thirty-five years later.

After the war he made up his mind to devote himself entirely to orthopaedics. In order to achieve his aim he took the unprecedented step of resigning his position as head of a surgical service in order to become assistant to P. Nové-Josserand. From this happy partnership came the first major French contribution to the surgery of bone tumours. Later, both with Nové-Josserand and independently, he studied the late results of treatment of congenital dislocation of the hip; and two of his pupils, Pierre Truchet and Albert Trillat, inspired by their master, made a detailed analysis of the results of certain operations used in the treatment of this condition, namely, subtrochanteric osteotomy, the shelf operation, and open reduction with which remodelling of the acetabulum was often combined. At about the same time Tavernier devised a simple method of reduction and fixation by grafting for fractures of the neck of the femur. He published an account of his own technique for the removal of damaged semilunar cartilages; he showed too a lively interest in recurrent dislocation of the shoulder and of the patella. At a time when the conservative treatment of joint tuberculosis was the commonly accepted practice in France he demonstrated and defended the merits of early arthrodesis for tuberculosis of the knee. He pointed out that central abscess of bone was a not infrequent sequel of osteomyelitis of childhood and of bone infection resulting from war wounds. Tavernier also described a painful apophysitis of the spine in some ways analogous to the epicondylar lesion of tennis elbow.

This summary of his work, though far from complete, is perhaps sufficient to give some indication of the fertility of Tavernier's labours between the two wars. The disaster of 1940 and the German occupation affected him profoundly. He sought refuge in his work and turned his mind to the awkward problem of the treatment of osteoarthritis of the hip. As is well known he was particularly concerned with endeavouring to relieve the condition by denervation of the joint. He found that anterior denervation, by section of the obturator nerve or of its sensory branches, gave poor results. He then concentrated on the posterior approach and found that resection of the nerve to quadratus femoris gave consistently better results. However, he was still far from content, and having reached the conclusion that the aim must be nothing less than total denervation he abandoned nerve section in favour of complete capsulectomy. Thus, after much hard work, he was convinced by the cold facts that much of his earlier reasoning had been misdirected and he was not afraid to say so. It was characteristic of Tavernier that he was as ready to admit defeat as to claim what, at first, had every appearance of being success.

This episode, late in his career, is some indication of the character of this great surgeon. Those who knew him well will always recall to mind a man of immense vitality who aroused enthusiasm in everyone he met. Tavernier was a great *chef de service*, never brushing aside a difficult question or an awkward critic; always more severe with himself than with others, he carried conviction by the sincerity and depth of his reasoning. When stricken at last by a progressive paralysis that confined him to his bed for the last eight months of his life his spiritual strength enabled him to conceal much of his suffering from those about him. In Louis Tavernier the venerable Société de Chirurgie de Lyon has lost the quickening influence of its greatest elder statesman; and France has lost an incomparable teacher, a beloved surgeon, and a great gentleman. A. T.

PROCEEDINGS AND REPORTS OF UNIVERSITIES, COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN

JOINT MEETING OF THE ORTHOPAEDIC ASSOCIATIONS OF THE ENGLISH-SPEAKING WORLD, 1952

In the first week of July 1952, from Sunday, June 29, to Friday, July 4, there will be held in London a joint meeting of all Orthopaedic Associations of the English-speaking world—of Britain, America, Canada, Australia, New Zealand, South Africa, and the other Dominions and Colonies of the Commonwealth of Nations, together with visitors from other countries.

On Sunday our visitors will be welcomed in the hotels of London. In the evening, at Claridges, there will be an official reception for all surgeons and their wives by Sir Reginald and Lady Watson-Jones, representing the British Orthopaedic Association. On this occasion it is hoped that British fellows and members will complete their arrangements for the entertainment of visiting guests.

The Inaugural Ceremony will be in the Beveridge Hall, Senate House, University of London, at 11 a.m. on the morning of Monday, June 30. Registration should be completed by then; indeed it is hoped that every fellow and member will have completed his registration before April 1. If a form of registration has not already been received it is by postal error and members of the Associations should write at once to Miss Key, Joint Secretariat, Lincoln's Inn Fields, London, W.C. 2, or to Miss Robertson, Congress Secretary, 82 Portland Place, London, W. 1.

Her Majesty the Queen hopes to attend the Inaugural Ceremony and decorate the presidents of visiting Orthopaedic Associations with jewels of office presented by fellows and members of the British Orthopaedic Association in the trust that these presidential badges will be worn in perpetuity at all future meetings of the respective Associations. The ceremony will be attended, in their academic robes and with insignia of office, by the Chancellor of the University of London, the Earl of Athlone, K.G., G.C.B., G.C.M.G., G.C.V.O., P.C., D.S.O., and Her Royal Highness the Princess Alice, G.C.V.O., G.B.E.; by the Principal of the University, Dr D. W. Logan, D.Ph., M.A., the Vice-Chancellor, Professor Bellot, M.A., and the deputy Vice-Chancellor, Mr P. H. Mitchiner, F.R.C.S.; by the President of the Royal College of Surgeons of England, Sir Cecil Wakeley; the President of the Royal College of Physicians of London, Sir Walter Russell Brain; the President of the Royal College of Surgeons of Edinburgh, Professor Walter Mercer; the President of the Royal Society of Medicine, Lord Webb-Johnson; other representatives of the Colleges of England, Scotland and Ireland; and by Lord Nuffield, Sir Arthur Sims and other distinguished guests.

Papers will be presented and discussions held in the Beveridge Hall of the University in the afternoon of Monday, June 30, and on the mornings of Tuesday, Wednesday, Thursday and Friday. There will be two clinical demonstrations, one at the Country Branch of the Royal National Orthopaedic Hospital on Wednesday and one at various London hospitals on Friday. The other afternoons will be free and it is hoped that British fellows and members will plan to entertain their guests on these days.

Two statutory lectures of the Royal College of Surgeons of England are being delivered during the meeting: the Robert Jones Lecture by Sir Max Page, K.B.E., on Tuesday evening; and a Moynihan Lecture on Thursday evening; and on these days there will be demonstrations of historical interest in the College.

Members of the Robert Jones Clubs of Britain and America, and members of the A.B.C. Club as well as Old Oswestrians and Old M.Ch.Orthians are planning special dinners during the week. There will also be many private dinners, cocktail parties and entertainments arranged by British fellows and members on Sunday, Monday, Wednesday and Friday.

On Tuesday we shall all go to the Ballet performance at the Royal Opera House, Covent Garden; on Thursday there will be the official Annual Dinner at the Dorchester; and on Friday, after private dinners and cocktail parties we shall relax with a ball, cabaret and fireworks displays at Hurlingham Club.

This photograph by Adams, Holden and Pearson is reproduced by permission of the University of London

The joint meeting of the British, American, Canadian, Australian, New Zealand and South African Orthopaedic Associations, together with surgeons from other parts of the Commonwealth and visitors from other countries, will be in the Senate House of the University of London in the first week of July—from Sunday, June 29, to Friday, July 4.

OFFICERS OF THE ORTHOPAEDIC ASSOCIATIONS OF THE BRITISH COMMONWEALTH AND THE UNITED STATES OF AMERICA

GREAT BRITAIN

President—Sir Reginald Watson-Jones, 82 Portland Place, London, W. 1.
Secretary—P. H. Newman, Esq., D.S.O., M.C., 45 Lincoln's Inn Fields, London, W.C. 2.

UNITED STATES OF AMERICA

President—Dr Fremont Chandler, 6 North Michigan Avenue, Chicago, Illinois, U.S.A.
Secretary—Dr C. Leslie Mitchell, Henry Ford Hospital, Detroit, Michigan, U.S.A.

CANADA

President—Dr R. Graham Huckell, 916 McLeod Buildings, Edmonton, Alberta, Canada.
Secretary—Dr W. B. Mackinnon, Canadian Orthopaedic Association, 661 Broadway Avenue, Winnipeg, Manitoba, Canada.

AUSTRALIA

President—Esmond F. West, Esq., Goldsbrough House, 172 North Terrace, Adelaide, Australia.
Secretary—Alex. R. Hamilton, Esq., 135 Macquarie Street, Sydney, New South Wales, Australia.

NEW ZEALAND

President—Alex Gillies, Esq., Masonic Chambers, 38 The Terrace, Wellington, New Zealand.
Secretary—J. Kennedy Elliott, Esq, 91 The Terrace, Wellington, New Zealand.

SOUTH AFRICA

President—G. T. du Toit, Esq., Orthopaedic Building, 79 de Villiers Strasse, Johannesburg.
Secretary—W. T. Ross, Esq., Orthopaedic Building, 79 de Villiers Strasse, Johannesburg.

ORGANISING COMMITTEE IN GREAT BRITAIN

Sir Reginald Watson-Jones (*President*)
C. Gordon Irwin (*Vice-President*)
S. L. Higgs (*Past President*)
George Perkins (*Former President*)
V. H. Ellis (*Treasurer*)
P. H. Newman (*Secretary*)
J. I. P. James (*Programme Secretary*)
B. H. Burns
Bryan McFarland
Walter Mercer
H. Osmond-Clarke
H. J. Seddon
Philip Wiles

Joint Secretariat—British Orthopaedic Association

Miss B. M. Key, 45 Lincoln's Inn Fields, London, W.C. 1

Secretary of Congress

Miss M. W. Robertson, 82 Portland Place, London, W. 1

LADIES COMMITTEE

Lady Watson-Jones
The Hon. Mrs Burns
Mrs Coltart
Lady Fairbank
Mrs Law
Mrs McFarland
Mrs Newman
Mrs Nissen
Mrs Osmond-Clarke
Mrs Seddon
Mrs Wiles

PROVISIONAL PROGRAMME OF THE JOINT MEETING OF ORTHOPAEDIC ASSOCIATIONS OF THE ENGLISH-SPEAKING WORLD

JUNE 29 TO JULY 4

	MORNING	AFTERNOON	EVENING
Sunday June 29	Reception at Hotels		5.30 Reception by Sir Reginald and Lady Watson-Jones at Claridges
Monday June 30	Registration at the University of London 11 a.m. Inaugural Ceremony H.M. the Queen	2.30 Scientific Programme London University	7.30 Private Dinners: Old Oswestrians. M.Ch.Orthians Savoy Hotel
Tuesday July 1	9.30 Scientific Programme London University	Free afternoon 5.00 Robert Jones Lecture Royal College of Surgeons	7.00 Ballet at Royal Opera House Covent Garden
Wednesday July 2	9.30 Scientific Programme London University	2.30 Clinical Demonstration Stanmore	7.30 Private Dinners: Robert Jones Clubs. Traveller's Club, Pall Mall A.B.C. Club Dinner Savoy Hotel
Thursday July 3	9.30 Scientific Programme London University	Free afternoon 5.00 Moynihan Lecture Royal College of Surgeons	7.00 Official Dinner at Dorchester Hotel
Friday July 4	9.30 Scientific Programme London University	2.30 Clinical Demonstrations at London Hospitals	9.30 July 4 Celebration at Hurlingham Club

VISITS TO ORTHOPAEDIC CENTRES IN GREAT BRITAIN AFTER THE JOINT MEETING IN THE FIRST WEEK OF JULY 1952

After the official arrangements in London there will be many orthopaedic clinical meetings arranged in centres throughout the country. A detailed list of the routine programmes in various British centres is tabulated in pages 135 to 147, and it is hoped that visiting surgeons, as well as surgeons at home, will plan to spend a week or two seeing the work in a single clinic. There is no doubt that this is the best way to learn the trends of orthopaedic surgery. Many think that there is far greater benefit in spending two or three weeks in a single clinic than in chasing for a day or so from one clinic to the next. Others may wish to visit a number of clinics in a short time and for them we will try to arrange tours, for example: Exeter, Bath, Bristol and Oxford; Norwich, Cambridge, Mansfield and Nottingham; Oswestry, Liverpool, Manchester, Edinburgh; and so on.

Whatever plans you may make you may be sure that there will be special invitations which will soon be published in detail. There are special programmes by Mr Capener in Exeter, and Mr Scott and Professor Trueta in Oxford. Mr Brittain has a particular programme in Norwich over the week-end immediately after the meeting, and you could go on to Cambridge with Mr Butler and Mr Fairbank, and to Nottingham and Mansfield with Mr Malkin and Mr Nicoll. There will be a special programme at the Robert Jones and Agnes Hunt Orthopaedic Hospital on Monday and Tuesday, July 7 and 8; and one in Liverpool and Manchester on Wednesday and Thursday of that week. Professor Mercer, president of the Royal College of Surgeons of Edinburgh, is arranging a special programme on Friday, July 9, with entertainment in his College and elsewhere on Saturday, July 10. Thereafter you could go fishing, or exploring the Lochs, or go back to the Lake District or to North Wales. There will be so much to do that you will be far too busy. Meanwhile here is a list of all the orthopaedic activities in Great Britain in the weeks after the official meeting in the first week of July.

TIME-TABLE OF CLINICAL PROGRAMMES IN LONDON AND PROVINCIAL CENTRES

It will be appreciated that in some cases this time-table covers only a part of the activities of the surgeons in the various hospitals listed. There are other sessions that visitors would be likely to find unprofitable either because the pressure of work is great, or because the needs of undergraduates must receive first consideration, or because the quarters in which the sessions are held are uncomfortably small.

It would be appreciated if intending visitors would communicate in advance with the surgeons or their secretaries so that they may be told which particular sessions are likely to be of special interest to them.

LONDON

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
Charing X Hospital Mr D. Trevor	Mon. Wed.	10.00 a.m. 9.30 a.m.	Mr D. Trevor Mr D. Trevor	Fri.	11.30 a.m.	Mr D. Trevor	Thu. Fri.	9.30 a.m. 9.30 a.m.	Mr D. Trevor Fracture Clinic
Guy's Hospital Mr T. T. Stamm							Mon. Mon. Wed. Fri.	2.00 p.m. 2.00 p.m. 2.00 p.m. 2.00 p.m.	Mr J. S. Batchelor Mr T. T. Stamm (Fracture Clinic) Mr T. T. Stamm Mr J. S. Batchelor (Fracture Clinic)
Orpington Hospital Mr T. T. Stamm	Wed. Thu.	9.30 a.m. 9.30 a.m.	Mr J. S. Batchelor Mr T. T. Stamm						
Hospital for Sick Children Great Ormond Street Mr E. I. Lloyd							Mon. Tue. Thu.	10.30 a.m. 10.30 a.m. 10.30 a.m.	Mr E. I. Lloyd Mr Denis Browne (Club Foot) Mr Denis Browne
King's College Hospital Mr St J. D. Buxton	Thu. Fri.	2.00 p.m 2.00 p.m.	Mr H. L.-C. Wood Mr St J. D. Buxton	Tue.	10.00 a.m.	Mr St J. D. Buxton and Mr H. L.-C. Wood	Mon. Tue. Wed.	9.30 a.m 9.30 a.m. 2.00 p.m.	Mr H. L.-C. Wood Fracture Clinic Mr St J. D. Buxton and Mr H. L.-C. Wood
London Hospital Sir R. Watson-Jones	Mon. Thu.	2.00 p.m. 2.00 p.m.	Mr O. Vaughan-Jackson Sir R. Watson-Jones, Mr H. Osmond-Clarke or Mr Law	Tue.	11.00 a.m.	Staff Conference	Tue.	2.00 p.m.	Undergraduate Teaching
Luton and Dunstable Hospital Mr L. W. Plewes						Invitation to Hand Infection Unit and Rehabilitation Centre by Arrangement			

LONDON

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
Middlesex Hospital Mr P. Wiles	Thu. Thu.	9.00 a.m. 2.00 p.m.	Mr P. H. Newman Mr P. Wiles	Wed.	2.00 p.m.	Joint Teaching Session. Grand Round	Tue. Wed. Fri. Sat.	10.00 a.m. 10.00 a.m. 10.00 a.m. 10.00 a.m.	Mr P. Wiles Mr P. Wiles (Fracture Clinic) Mr P. H. Newman Mr P. H. Newman (Fracture Clinic)
National Spinal Injuries Centre, Stoke Mandeville, Aylesbury, Bucks				Mon. Tue.	9.30 a.m. 9.30 a.m.	Dr L. Guttmann Dr L. Guttmann	Wed.	2.00 p.m.	Lecture Demonstrations, Dr L. Guttmann
Royal National Orthopaedic Hospital, Great Portland St., London, W. 1 Mr H. J. Seddon	Mon. Mon. Tue. Wed. Wed. Thu. Thu. Fri. Fri.	9.30 a.m. 2.00 p.m. 2.00 p.m. 9.30 a.m. 2.00 p.m. 9.30 a.m. 2.00 p.m. 9.30 a.m. 2.00 p.m.	Mr R. Y. Paton Mr J. I. P. James Mr K. I. Nissen Mr V. H. Ellis Mr D. Trevor Mr H. J. Seddon Mr H. J. Seddon Mr Jackson Burrows Mr A. T. Fripp	Wed. Thu.	9.30 a.m. 9.30 a.m.	Mr H. J. Seddon and Mr J. I. P. James Mr V. H. Ellis and Mr Jackson Burrows	Mon. Mon. Mon. Mon. Mon. Tue. Wed. Thu. Thu. Fri. Fri. Fri.	9.30 a.m. 9.30 a.m. 9.30 a.m. 2.00 p.m. 2.00 p.m. 2.00 p.m. 2.00 p.m. 9.30 a.m. 2.00 p.m. 10.00 a.m. 9.30 a.m. 2.00 p.m.	Mr H. J. Seddon and Mr D. Brooks (Nerve Injuries) Mr J. I. P. James (Hand Injuries) Mr P. H. Newman (Spinal Disorders) Mr D. Trevor (Congenital Dislocation of the Hip) Mr K. I. Nissen Mr A. T. Fripp, and Mr P. H. Newman Mr H. J. Seddon and Mr J. I. P. James (Congenital Dislocation of the Hip. 2nd Wednesday) Mr J. I. P. James (Scoliosis) Mr R. Y. Paton and Mr J. A. Cholmeley Mr A. T. Fripp (Club Foot) Mr K. I. Nissen and Mr J. I. P. James (Limb Equalisation) Mr V. H. Ellis and Mr Jackson Burrows
Royal National Orthopaedic Hospital, Brockley Hill, Stanmore Mr H. J. Seddon	Mon. Tue. Tue. Wed. Wed. Thu. Fri. Fri. Sat.	2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m.	Mr P. H. Newman Mr H. J. Seddon and Mr J. I. P. James Mr R. Y. Paton Mr A. T. Fripp Mr K. I. Nissen Mr J. A. Cholmeley Mr D. Trevor Mr D. N. Matthews	Tue. Tue. Wed Thu. Fri. Fri	9.30 a.m 10.00 a.m. 9.15 a.m. 2.00 p.m. 9.15 a.m. 2.00 p.m.	Mr D. Trevor (Congenital Dislocation of the Hip). Alternate weeks Mr K. I. Nissen Mr A. T. Fripp, Mr R. Y. Paton and Mr J. A. Cholmeley Mr P. H. Newman Mr H. J. Seddon Mr J. I. P. James			

QUEEN MARY'S HOSPITAL FOR CHILDREN, Carshalton Mr R. H. METCALFE	Wed Fri.	9.30 a.m. 9.30 a.m.	Mr R. G. Thomas Mr R. H. Metcalfe	Mon. Tue. Thu. Fri. Fri.	9.30 a.m. 2.00 p.m. 2.00 p.m. 9.30 a.m. 2.00 p.m.	Mr R. H. Metcalfe Mr R. G. Thomas Mr R. G. Thomas Mr R. G. Thomas Mr R. G. Thomas	Mon. Thu. Thu.	9.30 a.m. 9.30 a.m. 2.00 p.m.	Mr R. G. Thomas Mr R. H. Metcalfe Poliomyelitis Conference (Alternate Thursdays)
QUEEN MARY'S HOSPITAL, ROEHAMPTON Ministry of Pensions Mr ST J. D. BUXTON							Wed.	10.00 a.m.	Consultation and Visit to Limb Fitting Centre
ROYAL NORTHERN HOSPITAL Mr E. LLOYD	Wed. Wed.	9.30 a.m. 2.00 p.m.	Minor Operations and Manipulations	Mon.	9.30 a.m.		Mon. Tue. Thur.	9.30 a.m. 9.30 a.m. 9.30 a.m.	Fracture Clinic Registrar Orthopaedic Out-patients, Fracture Clinic—Registrar, Rheumatism Clinic
ST BARTHOLOMEW'S HOSPITAL Mr S. L. HIGGS							Mon. Thu.	1.30 p.m. 1.30 p.m.	Mr Jackson Burrows Mr S. L. Higgs & Mr W. D. Coltart
HILL END HOSPITAL, ST ALBANS Mr S. L. HIGGS	Mon. Fri.	10.00 a.m. 10.00 a.m.	Mr W. D. Coltart Mr W. D. Coltart	Mon. Tue. Tue.	2.30 p.m. 10.00 a.m. 2.00 p.m.	Mr W. D. Coltart Mr Jackson Burrows Mr S. L. Higgs			
ST GEORGE'S HOSPITAL Mr B. H. BURNS	Thu.	9.30 a.m.	Mr B. H. Burns and Mr R. H. Young						
ST THOMAS'S HOSPITAL Professor G. PERKINS	Mon. Thu.	2.00 p.m. 10.00 a.m.	Mr R. J. Furlong Mr R. J. Furlong	Thu.	9.00 a.m.	Professor G. Perkins and Mr R. J. Furlong	Tue. Thu.	2.00 p.m. 2.00 p.m.	Mr R. G. Furlong (Undergraduate Teaching) Mr R. G. Furlong
THE ROYAL FREE HOSPITAL Mr C. GRAY	Tue.	2.00 p.m.	Mr C. Gray	Fri.	11.00 a.m.	Mr C. Gray	Fri.	2.00 p.m.	Mr C. Gray
UNIVERSITY COLLEGE HOSPITAL Mr C. FLEMMING							Thu.	2.30 p.m.	Mr C. Flemming
WESTMINSTER HOSPITAL	Thu.	2.00 p.m.	Mr E. P. Brockman and Mr H. E. Harding				Mon. Wed. Fri.	10.00 a.m. 10.00 a.m. 10.00 a.m.	Mr E. P. Brockman and Mr H. E. Harding Mr E. P. Brockman and Mr H. E. Harding Mr E. P. Brockman

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
ABERDEEN									
Aberdeen Royal Infirmary Mr A. M. Rennie	Mon. Thu.	9.00 a.m. 9.00 a.m.	Mr A. M. Rennie Mr N. G. C. Hendry	Sat.	10.00 a.m.	Mr A. M. Rennie	Mon. Tue. Thu. Fri.	9.30 a.m. 9.30 a.m. 2.00 p.m. 9.30 a.m.	Mr Alexander Adam Mr N. G. C. Hendry Mr G. Hay Mr A. M. Rennie and Mr N. G. C. Hendry
Royal Aberdeen Hospital for Sick Children	Wed.	2.15 p.m.	Mr A. M. Rennie				Wed.	10.30 a.m.	Mr A. M. Rennie and Mr N. G. C. Hendry
Strathcaro Hospital, Brechin	Wed. Fri.	9.00 a.m. 9.00 a.m.	Mr G. Hay Mr G. Hay	Tue.	10.00 a.m.	Mr A. M. Rennie			
ALTON									
Lord Mayor Treolar Orthopaedic Hospital Mr E. S. Evans	Wed. Thu. Fri.	10.00 a.m 9.00 a.m. 9.00 a.m.	Mr E. W. Peet (Plastic) Mr V. H. Ellis or Mr H. H. Langston Mr E. S. Evans	Mon. Thu.	10.30 a.m. July 10 11.00 a.m.	Mr E. S. Evans Orthopaedic Conference Sir Thomas Fairbank & Consulting Surgeons	Mon. Wed.	2.00 p.m. 10.00 a.m.	Mr J. D. Wilson Mr E. W. Peet (Plastic)
BATH									
Bath and Wessex Orthopaedic Hospital Mr J. Bastow	Mon. Tue. Wed. Thu. Fri.	9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m.	Mr M. H. Hall Mr A. E. Burton Mr J. Bastow Mr T. Price Mr J. Bastow						
Royal United Hospital				Sun.	10.30 a.m.	Mr J. Bastow and Mr A. E. Burton	Mon. Thu.	2.15 p.m. 2.15 p.m.	Mr J. Bastow, Mr A. E. Burton and Mr M. H. Hall (Fracture Clinic) Mr J. Bastow, Mr A. E. Burton and Mr Hughes-Davies (Fracture Clinic)
Royal National Hospital for Rheumatic Diseases				Tue.	2.15 p.m.	Mr J. Bastow and Mr A. E. Burton			

BELFAST									
Royal Victoria Hospital Mr R. J. W. Withers	Mon.	9.30 a.m.	Mr R. J. W. Withers	Thu.	9.30 a.m.	Mr R. J. W. Withers	Tue. Wed. Fri.	2.00 p.m. 9.30 a.m. 9.30 a.m.	Mr R. J. W. Withers (Fracture Clinic) Mr R. J. W. Withers and Mr R. I. Wilson Mr N. S. Martin
Musgrave Park Hospital	Mon. Tue. Thu.	9.30 a.m. 9.30 a.m. 9.30 a.m.	Mr R. I. Wilson Mr R. J. W. Withers Mr N. S. Martin	Wed. Fri. Fri.	2.00 p.m. 9.30 a.m. 2.00 p.m.	Mr N. S. Martin Mr R. J. W. Withers Mr R. I. Wilson			
BIRMINGHAM									
Royal Orthopaedic Hospital 80 Broad St. (Out-patients) Mr F. G. Allan							Tue. Fri.	9.00 a.m. 9.00 a.m.	Mr F. G. Allan Mr T. S. Donovan
Woodland Hospital, Northfield (In-patients)	Mon. Thu. Fri. Fri. (After Inde)	9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m.	Mr F. G. Allan Mr T. S. Donovan Mr A. M. Hendry Mr F. G. Allan	Tue.	2.30 p.m.	Mr A. M. Hendry			
Children's Hospital	Wed.		Mr F. G. Allan				Wed.		Mr F. G. Allan
Birmingham Accident Hospital	Daily operating sessions, rounds and clinics and an emergency service that goes on over the twenty-four hours								
BRADFORD									
Bradford Royal Infirmary Mr A. Naylor	Tue. Thu.	9.00 a.m. 9.00 a.m.	Mr J. Wishart Mr A. Naylor	Tue. Sat.	1.30 p.m. 9.00 a.m.	Mr A. Naylor Mr J. Wishart	Mon. Tue. Wed. Thu.	2.00 p.m. 2.00 p.m. 2.00 p.m. 2.00 p.m.	Mr J. Wishart (Follow-up Clinic) Mr A. Naylor (Follow-up Clinic) Mr J. Wishart Mr A. Naylor
BRISTOL									
Bristol Royal Infirmary Mr A. L. Eyre-Brook	Mon. Tue. Thu. Fri.	9.00 a.m. 2.00 p.m. 2.00 p.m. 2.00 p.m.	Mr M. P. McCormack Mr K. Lucas Mr K. Pridie Mr A. L. Eyre-Brook				Mon. Mon. Tue. Tue. Wed. Wed. Thu. Thu. Fri. Fri. Sat.	9.00 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m. 9.00 a.m.	Mr K. Pridie Mr K. Lucas Mr K. Pridie Mr M. P. McCormack Mr A. L. Eyre-Brook Mr K. Lucas Mr A. L. Eyre-Brook Mr M. P. McCormack Mr K. Pridie Mr K. Lucas Mr M. P. McCormack
Winford Orthopaedic Hospital	Tue. Wed.	All day All day	Mr A. L. Eyre-Brook Mr K. Pridie	Wed. Sat.	9.00 a.m.	Mr K. Pridie Mr A. L. Eyre-Brook			

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
CAMBRIDGE									
Addenbrooke's Hospital Mr R. W. Butler	Wed. Fri.	2.00 p.m. 2.00 p.m.	Mr R. W. Butler Mr T. J. Fairbank	Tue. Wed.	9.30 a.m. 9.30 a.m.	Mr R. W. Butler and Mr T. J. Fairbank	Mon. Wed. Thu. Fri.	2.00 p.m. 10.30 a.m. 2.00 p.m. 10.30 a.m.	Mr T. J. Fairbank (Fracture Clinic) Mr R. W. Butler and Mr T. J. Fairbank Mr T. J. Fairbank (Fracture Clinic) Mr R. W. Butler and Mr T. J. Fairbank
DERBY									
Derbyshire Royal Infirmary Mr R. G. Pulvertaft	Mon. Wed.	2.00 p.m. 9.15 a.m.	Mr E. W. O. Adkins Mr R. G. Pulvertaft	Fri.	10.00 a.m.	Mr R. G. Pulvertaft and Mr E. W. O. Adkins	Mon. Tue. Wed. Thu. Fri.	9.30 a.m. 9.30 a.m. 9.30 a.m. 9.30 a.m. 2.00 p.m.	Mr R. G. Pulvertaft Mr E. W. O. Adkins Mr E. W. O. Adkins (Back Clinic) Mr R. G. Pulvertaft (Hand Clinic) Mr E. W. O. Adkins (Orthopaedic and Fracture Clinic)
City Hospital	Thu.	2.00 p.m.	Mr E. W. O. Adkins						
Hospital for Sick Children	Mon.	2.15 p.m.	Mr R. Lunt				Thu.	2.00 p.m.	Mr R. Lunt
Harlow Wood Orthopaedic Hospital Mr S. A. S. Malkin	Tue. Thu. Thu. Fri. Fri. Sat.	2.00 p.m. 9.15 a.m. 2.00 p.m. 9.15 a.m. 2.00 p.m. 9.15 a.m.	Mr R. G. Pulvertaft (Hand Surgery) Mr S. A. S. Malkin Mr S. A. S. Malkin Mr J. P. Campbell Mr R. G. Pulvertaft (Hand Surgery) Mr J. P. Jackson	Mon. Tue.	10.00 a.m. 9.15 a.m.	Mr J. P. Campbell Mr S. A. S. Malkin	Fri.	11.00 a.m.	Mr J. P. Jackson (Rehabilitation Unit)
EDINBURGH									
Royal Infirmary Professor W. Mercer	Mon. Tue. Wed. Thu.	10.00 a.m. 2.00 p.m. 10.00 a.m. 10.00 a.m.	Mr I. L. Dick Mr E. A. Jack Mr I. L. Dick Mr E. A. Jack				Mon. Tue. Tue. Wed. Thu. Fri. Fri. Sat.	10.00 a.m. 10.00 a.m. 11.00 a.m. 10.00 a.m. 10.00 a.m. 10.00 a.m. 10.00 a.m. 10.00 a.m.	Mr E. A. Jack Mr I. L. Dick Mr E. A. Jack (Physiotherapy Clinic) Mr E. A. Jack Mr I. L. Dick Mr E. A. Jack Mr I. L. Dick (Physiotherapy Clinic) Mr I. L. Dick

Princess Margaret Rose Hospital, Fairmilehead	Wed. Thu. Fri.	10.00 a.m. 10.00 a.m. 2.30 p.m.	Mr W. V. Anderson Mr R. I. Stirling Mr R. I. Stirling	Mon. Tue.	10.00 a.m. 2.30 p.m.	Mr W. V. Anderson Mr R. I. Stirling	Mon Tue. Sat.	10.00 a.m. 2.30 p.m. 10.00 a.m.	Mr W. V. Anderson Mr R. I. Stirling Mr G. A. Pollock
Westerlea Spastic School							Mon. Sat.	2.30 p.m. 10.00 a.m.	Mr G. A. Pollock Mr G. A. Pollock
Sick Children's Hospital							Thu.	2.30 p.m.	Mr E. A. Jack or Mr I. L. Dick
Bangour Hospital							Thu.	2.30 p.m.	Mr E. A. Jack or Mr I. L. Dick (Surgical Tubercle Clinic)
EXETER The Princess Elizabeth Orthopaedic Hospital Mr N. L. CAPENER	Mon. Tue. Thu. Fri.	8.45 a.m. 8.45 a.m. 8.45 a.m. 8.45 a.m.	Mr N. L. Capener Mr G. Blundell-Jones Mr N. L. Capener Mr S. C. Durbin	Tue. Wed. Thu.	9.30 a.m. 9.30 a.m. 2.00 p.m.	Mr N. L. Capener Mr S. C. Durbin Mr G. Blundell-Jones	Tue. Thu.	2.00 p.m. 2.00 p.m.	Mr N. L. Capener Mr S. C. Durbin or Mr G. Blundell-Jones
GLASGOW Western Infirmary Mr R. BARNES	Tue Fri.	9.00 a.m. 9.00 a.m.	Mr T. W. Howat Mr J. Tulloch Brown	Mon. Thu.	9.00 a.m. 9.00 a.m.	Mr T. W. Howat Mr J. Tulloch Brown	Daily Mon. Wed. Thu. Fri.	9.00 a.m. 2.00 p.m. 2.00 p.m. 2.00 p.m. 2.00 p.m.	Fracture Clinic Mr A. R. Parkes (Hand and Peripheral Nerve Injuries) Mr R. Barnes and Mr J. Tulloch Brown Mr A. R. Parkes (Hand and Peripheral Nerve Injuries) Mr R. Barnes and Mr T. W. Howat
Victoria Infirmary	Mon. Thu.	9.00 a.m. 9.00 a.m.	Mr A. Miller and Mr T. H. Norton Mr T. H. Norton or Mr Watt				Mon. Wed. Wed.	2.00 p.m. 9.00 a.m. 2.00 p.m.	Mr A. Miller and Mr Watt Mr A. Miller and Mr T. H. Norton Mr T. H. Norton and Mr Watt
Killearn Hospital, Killearn	Mon. Wed. Thu.	9.00 a.m. 9.00 a.m. 9.00 a.m.	Mr R. Barnes and Mr J. Tulloch Brown Mr A. R. Parkes (Hand and Peripheral Nerve injuries) Mr R. Barnes and Mr T. W. Howat	Mon. Tue.	2.00 p.m. 10.00 a.m.	Mr R. Barnes and Mr Mr T. W. Howat Mr A. R. Parkes (Hand and Peripheral Nerve Injuries)			
Philipshill Hospital	Tue. Thu.	2.00 p.m. 9.00 a.m.	Mr T. H. Norton Mr A. Miller and Mr Watt				Tue.	9.00 a.m.	Mr A. Miller
Mearnskirk Hospital	Thu.	2.00 p.m.	Mr K. E. Guest						
Uddingston Rehabilitation Centre				Thu.	2.00 p.m.	Mr A. Miller	Daily		Rehabilitation and Medico-social Resettlement

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
HULL							Mon.	2.00 p.m.	Mr R. C. Tatham (Fracture Clinic)
							Tue.	2.30 p.m.	Mr R. C. Tatham (Fracture Clinic)
							Tue.	2.00 p.m.	Mr J. S. Maxwell
Hull Royal Infirmary	Mon.	2.00 p.m.	Mr J. S. Maxwell	Tue.	2.00 p.m.		Wed.	9.30 a.m.	Mr R. C. Tatham
Mr R. C. Tatham	Thu.	2.00 p.m.	Mr R. C. Tatham				Wed.	2.00 p.m.	Mr J. S. Maxwell (Fracture Clinic)
							Thu.	2.00 p.m.	Mr J. S. Maxwell (Fracture Clinic)
							Fri.	2.00 p.m.	Mr R. C. Tatham (Fracture Clinic)
Westwood Hospital	Tue.	10.00 a.m.	Mr J. S. Maxwell	Mon.	9.30 a.m.	Mr R. C. Tatham	Mon.		Mr R. C. Tatham
Beverley	Wed.	2.30 p.m.	Mr R. C. Tatham	Fri.	9.30 a.m.	Mr J. S. Maxwell	Fri.		Mr J. S. Maxwell
IPSWICH									
East Suffolk and Ipswich Hospital	Fri.	2.30 p.m.	Mr E. Bell-Jones or Mr K. Marks	Tue.	3.30 p.m.	Mr E. Bell-Jones or Mr K. Marks	Mon.	1.30 p.m.	Mr E. Bell-Jones or Mr K. Marks
Mr E. Bell-Jones	Sat.	9.30 a.m.					Thu.	1.30 p.m.	
Borough General Hospital	Mon.	9.30 a.m.	Mr E. Bell-Jones or Mr K. Marks	Fri.	9.30 a.m.	Mr E. Bell-Jones or Mr K. Marks	Wed.	2.00 p.m.	Mr E. Bell-Jones or Mr K. Marks
LEEDS							Mon.	2.00 p.m.	Registrar (Fracture Clinic)
							Tue.	9.00 a.m.	Mr R. Broomhead, Mr A. B. Pain and Mr J. P. M. Clark
							Tue.	2.00 p.m.	Registrar (Fracture Clinic)
							Wed.	9.00 a.m.	O.P. Operations—Registrar
							Wed.	2.00 p.m.	Mr R. Broomhead (Back Clinic)
							Wed.	2.00 p.m.	Resident Officer (Fracture Clinic)
	Mon.	9.00 a.m.	Mr R. Broomhead				Thu.	9.00 a.m.	Registrar (Fracture Clinic)
	Mon.	9.00 a.m.	Mr J. M. P. Clark				Thu.	9.00 a.m.	Mr A. B. Pain (Follow-up Clinic)
Leeds General Infirmary	Wed.	9.00 a.m.	Mr A. B. Pain						Mr R. Broomhead, Mr A. B. Pain
Mr R. Broomhead	Wed.	9.00 a.m.	Mr J. M. P. Clark				Thu.	2.00 p.m.	and Mr J. M. P. Clark (Follow-up Clinic)
	Fri.	2.00 p.m.	Mr R. Broomhead				Fri.	9.00 a.m.	Registrar (Fracture Clinic)
	Fri.	2.00 p.m.	Mr A. B. Pain				Sat.	9.00 a.m.	Registrar (Fracture Clinic)
							Sat.	9.00 a.m.	Mr J. M. P. Clark (Follow-up Clinic —Tendons)
							Sat.	9.00 a.m.	Mr A. B. Pain (Follow-up Clinic —Talipes)
							Sat.	9.00 a.m.	Manipulations and Plaster Jackets

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
LEEDS (*continued*)									
Royal Bath, Harrogate	Tue.	2.00 p.m.	Mr R. Broomhead				Sat.	9.30 a.m.	Mr R. Broomhead
Pinderfields Hospital (Poliomyelitis Unit)							Tue. Wed.	2.00 p.m. 2.00 p.m.	Mr J. M. P. Clark Mr J. M. P. Clark
LIVERPOOL									
Professor Bryan McFarland									
David Lewis Northern Hospital	Hospital Hours from 9.30 a.m. to 4 p.m. on Monday						Mr E. N. Wardle and Mr G. E. Thomas		
Walton Hospital, Liverpool	Hospital Hours from 9.30 a.m. to 4 p.m. on Monday						Mr P. B. Moroney		
Liverpool Royal Infirmary	Hospital Hours from 9.30 a.m. to 4 p.m. on Tuesday						Mr N. W. Roberts and Mr R. Roaf		
Royal Southern Hospital	Hospital Hours from 9.30 a.m. to 4 p.m. on Wednesday						Professor Bryan McFarland and Mr G. V. Osborne		
Royal Infirmary, Chester	Hospital Hours from 9.30 a.m. to 4 p.m. on Thurdsay						Mr G. T. Partridge		
Royal Liverpool Children's Hospital	4.00 p.m. Clinic on Thursday						Professor Bryan McFarland—Demonstrations only		
Leasowe Children's Hospital	Hospital Hours from 9.30 a.m. to 4 p.m. on Friday						Mr N. W. Roberts and Mr E. N. Wardle		
Heswall Children's Hospital	Hospital Hours from 9.30 a.m. to 4 p.m. on Friday						Professor Bryan McFarland and Mr G. E. Thomas		
Alder Hey Children's Hospital	Hospital Hours from 9.30 a.m. to 4 p.m. on Saturday						Mr F. C. Dwyer		

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
MANCHESTER							Daily		Fracture Clinics
Manchester Royal Infirmary Sir Harry Platt	Mon. Wed.	9.30 a.m. 9.30 a.m.	Sir Harry Platt Mr John Charnley	Tue.	10.00 a.m.	Staff	Tue. Thu.	2.00 p.m. 9.00 a.m.	Mr John Charnley Mr John Charnley (Fracture Clinic)
Biddulph Grange Orthopaedic Hospital	Wed. or Thu.		Sir Harry Platt				Wed. or Thu.		Sir Harry Platt (Clinical Demonstrations)
Wrightington Hospital Nr. Wigan	Mon.	2.00 p.m.	Mr John Charnley or Mr C. Cullen	Daily	9.30 a.m.	Dr J. Dobson			
Withington Hospital	Wed. Sat.	9.30 a.m. 9.30 a.m.	Mr E. S. Brentall Mr J. C. F. Cregan	Tue.	10.00 a.m.	Follow-up Cases	Tue.	2.00 p.m.	Fracture Clinic
Salford Royal Hospital	Mon. Fri.	9.30 a.m. 2.00 p.m.	Mr Sayle Creer or Mr D. D. Cranna Mr Sayle Creer				Wed. Daily	9.30 a.m.	Mr Sayle Creer, Mr D. D. Cranna or Mr M. L. Mangan Fracture Clinics
Royal Manchester Children's Hospital	Fri.	9.30 a.m.	Mr D. D. Cranna				Tue.	9.30 a.m.	Mr D. D. Cranna
Hope Hospital	Thu.	9.00 a.m.	Mr Sayle Creer or Mr D. D. Cranna				Mon.	2.00 p.m.	Mr Sayle Creer and Mr D. D. Cranna
Crumpsall Hospital	Mon. Thu.	9.30 a.m. 2.00 p.m.	Mr E. Flatow Mr D. Ll. Griffiths						
Booth Hall Hospital	Fri.	10.00 a.m.	Mr D. Ll. Griffiths						
Ancoats Hospital	Mon. Fri.	9.30 a.m. 9.30 a.m.	Mr D. Ll. Griffiths Mr E. Flatow				Tue. Thu.	2.00 p.m. 9.00 a.m.	Mr D. Ll. Griffiths and Mr E. Flatow Mr D. Ll. Griffiths and Mr E. Flatow (Major Fracture Clinic)
Park Hospital	Fri.	2.00 p.m.	Mr John Charnley	Thu.	2.00 p.m.	Mr John Charnley			
Eccles and Patricroft	Thu.	2.00 p.m.	Mr S. M. Milner				Tue.	9.00 a.m.	Mr S. M. Milner
Altrincham General Hospital	Thu. Sat.	9.30 a.m. 10.00 a.m.	Mr H. Poston Mr H. Poston				Tue.	9.30 a.m.	Mr H. Poston

NEWCASTLE Royal Victoria Hospital Mr C. Gordon Irwin	Mon. Tue. Wed. Fri.	9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m.	Registrar Mr C Gordon Irwin Mr J. K. Stanger Mr C. Gordon Irwin			Teaching Rounds three days a week	Mon. Tue. Wed Thu Thu. Fri.	9.30 a.m. 9.30 a.m. 9.30 a.m. 9.30 a.m. 2.00 p.m. 9.30 a.m.	Mr C. Gordon Irwin Mr J. K. Stranger Mr C. Gordon Irwin Mr J. V. Todd Mr A. E. Bremner Mr J. K. Stanger
NORWICH Norfolk and Norwich Hospital Mr H. A. Brittain	Mon. Tue. Wed. Thu. Thu. Fri. Fri.	8.30 a.m. 2.00 p.m. 9.00 a.m. 8.30 a.m. 2.00 p.m. 9.00 a.m. 2.00 p.m.	Mr G. K. McKee Registrar (Minor Operations) Mr H. A. Brittain Mr G. K. McKee Mr R. C. Howard Mr H. A. Brittain Mr R. C. Howard	Tue. Thu. Fri.	9.00 a.m. 9.00 a.m. 9.30 a.m.	Mr H. A. Brittain Mr R. C. Howard Mr G. K. McKee	Mon. Mon. Mon. Tue. Tue. Tue. Tue. Thu. Fri. Fri.	11.00 a.m. 12.00 a.m. 2.00 p.m. 9.30 a.m. 10.00 a.m. 10.30 a.m. 11.30 a.m. 10.00 a.m. 10.30 a.m. 2.30 p.m.	Mr H. A. Brittain Mr R. C. Howard Registrar (Manipulations) Mr G. K. McKee Mr R. C. Howard Mr H. A. Brittain Mr R. C. Howard (Massage Clinic) Mr R. C. Howard Mr G. K. McKee (Fracture Clinic) Mr H. A. Brittain and Mr G. K. McKee (Physiotherapy Clinic)
Jenny Lind Hospital for Children	Mon.	9.00 a.m.	Mr R. C. Howard				Tue. Wed. Wed. Thu. Sat.	2.30 p.m. 2.30 p.m. 2.30 p.m. 10.00 a.m. 10.00 a.m.	Mr G. K. McKee Mr H. A. Brittain Mr R. C. Howard Mr H. A. Brittain Mr R. C. Howard
Great Yarmouth Hospital	Mon. Wed.	3.00 p.m. 9.30 a.m.	Mr R. C. Howard Mr R. C. Howard				Mon.	3.00 p.m.	Mr H. A. Brittain
Melton Lodge Children's Orthopaedic Hospital				Mon.	5.15 p.m.	Mr H. A. Brittain			
Lowestoft Hospital	Tue.	4.30 p.m.	Mr H. A. Brittain				Tue.	3.00 p.m.	Mr H. A. Brittain
Cromer Hospital	Wed.	3.30 p.m.	Mr G. K. McKee				Wed.	2.30 p.m.	Mr G. K. McKee
OSWESTRY The Robert Jones and Agnes Hunt Orthopaedic Hospital Mr J. N. Wilson	Tue. Sat.	All day All day	Registrar Visiting Surgeon	Fri.	9.30 a.m.	Visiting Surgeon	Thu. Fri.	2.00 p.m. 9.30 a.m.	Registrar Visiting Surgeon
OXFORD Wingfield-Morris Orthopaedic Hospital Professor J. Trueta	Mon. Tue. Wed. Fri.	8.45 a.m. 8.30 a.m. 8.30 a.m. 8.30 a.m.	Mr J. C. Scott and Mr E. Somerville Mr R. G. Taylor Professor Trueta, Mr J. D. Morgan and Mr J. Agerholm Mr W. B. Foley	Mon. Mon. Mon. Wed. Wed. Fri.	9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m. 9.00 a.m.	Professorial Unit Mr W. B. Foley Mr J. Agerholm Mr R. G. Taylor Mr W. B. Foley Mr J. C. Scott and Mr E. Somerville	Wed. Thu. Thu. Sat. Sat.	10.30 a.m. 9.00 a.m. 10.30 a.m. 9.00 a.m. 10.00 a.m.	Mr R. G. Taylor and Mr E. Somerville Clinical Conference—Staff Professor Trueta, Mr W. B. Foley and Mr J. Agerholm Clinical Conference—Staff R.S.O. Minor Operations
Radcliffe Infirmary	Wed. Fri.	2.00 p.m. 2.00 p.m.	Mr J. C. Scott Mr R. G. Taylor	Mon. Thu.	2.00 p.m. 2.00 p.m.	Mr J. C. Scott Mr R. G. Taylor	Daily	10.00 a.m.	Mr J. C. Scott and Mr R. G. Taylor (Fracture Clinic)

PROVINCIAL CENTRES

Hospital	Operating Sessions			Ward Rounds			Out-patients		
	Day	Time	Details	Day	Time	Details	Day	Time	Details
PLYMOUTH									
Mount Gold Orthopaedic Hospital Mr G. J. Lillie	Mon. Tue. Thu. Fri.	9.00 a.m. 9.00 a.m. 9.00 a.m. 9.30 a.m.	Mr G. J. Lillie Mr M. H. Salz Mr G. J. Lillie Mr M. H. Salz	Mon. Tue.	9.00 a.m. 9.00 a.m.	Mr M. H. Salz Mr G. J. Lillie	Tue. Thu. Thu. Fri. Fri.	2.00 p.m. 9.00 a.m. 2.00 p.m. 9.30 a.m. 2.00 p.m.	Mr G. J. Lillie Mr M. H. Salz Mr M. H. Salz Mr G. J. Lillie (Peripheral Clinic) Mr M. H. Salz (School Clinic)
South Devon and East Cornwall Hospital	Wed. Sat.	9.00 a.m. 9.00 a.m.	Mr M. H. Salz Mr G. J. Lillie and Mr M. H. Salz	Wed.	9.00 a.m.	Mr G. J. Lillie	Mon. Tue. Wed. Thu.	2.00 p.m. 2.00 p.m. 2.00 p.m. 2.00 p.m.	Mr M. H. Salz (Fracture Clinic) Mr M. H. Salz (Fracture Clinic) Mr G. J. Lillie and Mr M. H. Salz Mr G. J. Lillie (Fracture Clinic)
Liskeard Clinic							Thu.	10.00 a.m.	Mr G. J. Lillie (Peripheral Clinic)
Kingsbridge Clinic							Tue.	9.30 a.m.	Mr M. H. Salz (Peripheral Clinic)
SHEFFIELD									
Royal Infirmary Mr F. Holdsworth	Mon. Wed.	2.00 p.m.	Mr A. Dornan Mr F. Holdsworth	Mon. Fri.	2.00 p.m. 9.30 a.m.	Mr F. Holdsworth Mr F. Holdsworth	Daily except Wed.		Mr F. Holdsworth (Fracture Clinic)
Firbeck Rehabilitation Centre							Thu.	2.00 p.m.	Mr F. Holdsworth
Royal Hospital	Thu.	2.00 p.m.	Mr A. Dornan				Wed.	9.30 a.m.	Mr A. Dornan
City General Hospital	Tue. Thu.	2.30 p.m. 9.30 a.m.	Mr S. Pappworth Mr S. Pappworth	Mon. Wed. Fri.	2.15 p.m. 2.15 p.m. 2.15 p.m.	Mr S. Pappworth Mr S. Pappworth Mr S. Pappworth	Mon. Wed. Fri.	9.30 a.m. 9.30 a.m. 9.30 a.m.	Mr S. Pappworth Mr S. Pappworth Mr S. Pappworth

SOUTHAMPTON									
Royal South Hants and Southampton Hospital Mr H. H. LANGSTON	Wed. Wed.	9.30 a.m. 2.00 p.m.	Mr J. S. Ellis Mr H. H. Langston	Tue.	11.00 a.m.	All Surgeons	Mon. Mon. Tue. Tue. Wed. Wed. Thu. Fri. Sat.	9.30 a.m. 2.00 p.m. 9.30 a.m. 2.00 p.m. 10.30 a.m. 2.00 p.m. 10.30 a.m. 10.00 a.m. 9.30 a.m.	Mr J. S. Ellis (Hand Clinic and Fracture Clinic) Mr J. S. Ellis Mr R. Bloom (Fracture Clinic) Mr R. Bloom Mr H. H. Langston (Children's Clinic) Mr R. Bloom (Fracture Clinic) Mr R. Bloom (Fracture Clinic) Mr H. H. Langston Mr R. Bloom or Mr J. S. Ellis
Royal Hants County, Winchester	Tue. Wed. Thu.	2.00 p.m. 2.00 p.m. 9.30 a.m.	Mr H. H. Langston Mr J. S. Ellis Mr J. S. Ellis	Tue.	9.30 a.m.	Mr H. H. Langston and Mr J. S. Ellis	Mon. Mon. Thu.	11.00 a.m. 2.00 p.m. 2.00 p.m.	Mr H. H. Langston (Fracture Clinic) Mr H. H. Langston Mr J. S. Ellis (Fracture Clinic)
General Infirmary, Salisbury	Thu. Thu.	10.00 a.m. 2.00 p.m.	Mr J. B. Shield Mr J. B. Shield	Mon.	9.30 a.m.	Mr J. B. Shield	Mon. Wed. Wed. Wed. Fri.	2.00 p.m. 9.30 a.m. 9.30 a.m. 2.00 p.m. 9.30 a.m.	Mr J. B. Shield (Fracture Clinic) Mr J. B. Shield Mr H. H. Langston (Children's) Clinic) Mr J. B. Shield (Fracture Clinic) Mr J. B. Shield (Fracture Clinic)
Odstock Hospital and General Infirmary				Mon. Fri.	9.30 a.m. 2.00 p.m.	Mr J. B. Shield Mr J. B. Shield			
YORK									
County Hospital Mr T. J. B. A. MACGOWAN	Wed. Fri.	2.00 p.m. 9.15 a.m.	MrT. J. B. A. MacGowan MrT. J. B. A. MacGowan				Mon. Tue. Wed. Thu. Sat.	9.15 a.m. 2.00 p.m. 9.15 a.m. 2.00 p.m. 9.15 a.m.	Mr T. J. B. A. MacGowan Mr T. J. B. A. MacGowan Mr T. J. B. A. MacGowan Mr T. J. B. A. MacGowan Mr T. J. B. A. MacGowan
City Hospital	Tue. Thu.	9.15 a.m. 2.15 p.m.	Mr D. V. Marshall Mr D. V. Marshall				Tue. Fri.	2.00 p.m. 2.00 p.m.	Mr D. V. Marshall Mr D. V. Matshall
Poppleton Annexes				Sat.	9.30 a.m.	Mr D. V. Marshall			

BRITISH ORTHOPAEDIC ASSOCIATION

ANNUAL MEETING 1951

The annual meeting of the British Orthopaedic Association was held in Edinburgh on October 25, 26 and 27, 1951, under the presidency of Mr S. L. Higgs.

SYMPOSIUM ON THE MANAGEMENT OF RHEUMATOID ARTHRITIS

The president of the association, Mr S. L. Higgs, introducing the first speaker, said that he had felt concern over the lack of co-operation between physicians and surgeons in the treatment of patients with rheumatoid arthritis. It was common to see patients in medical wards with established deformities which could have been prevented by early orthopaedic care. Physicians often failed to realise what could be done to prevent deformity; and orthopaedic surgeons did not always appreciate sufficiently the help that could be given by their medical colleagues. It was for these reasons that he had suggested this symposium, which he hoped would foster a spirit of closer co-operation in the future.

Recent advances in medical treatment—*Dr J. R. Duthie* (Edinburgh) said that the introduction of cortisone and A.C.T.H. had tended to obscure other advances that had been made, but these were nevertheless important. A few years ago the hypothesis of focal sepsis as an important etiological factor was widely accepted. Rheumatoid arthritis was regarded as essentially a constitutional disease and it was believed that treatment should be directed primarily towards encouraging constitutional improvement. A wide variety of illogical methods of treatment were suggested and practised, but too little attention was paid to the condition of the joints themselves, which, though inflamed, were subjected to daily passive movements. One of the most important recent advances had been the abandonment of this "uncontrolled empiricism" in the management of the disease. Controlled investigations were now being carried out, and they had already led to the discarding of many remedies for which extravagant and unfounded claims had been made. The only remedy that had survived was gold, and its reputation had suffered severely.

It was possible, he submitted, that the constitutional disturbance of rheumatoid arthritis was secondary to the severe inflammatory changes in the joints, rather than the reverse. With this possibility in mind he was investigating the results of resting the affected joints rather than submitting them to passive movements. The limb was rested in a plaster splint and the muscles were exercised by static contractions. Pain was rapidly relieved in most cases, and the need for analgesics was reduced. The inflammation of the joints appeared to settle relatively quickly and ankylosis had not occurred. After the period of rest, which lasted a few weeks, an attempt was made by resistance exercises to restore joint stability before weight-bearing was allowed.

A notable advance in the approach to the problem of rheumatoid arthritis had been the establishment of special units in which full co-operation between different specialists was possible. Segregation of patients in centres such as these was often criticised, but it had the great advantages for the patient that it facilitated efficient treatment and adequate follow-up examination.

Referring to Cortisone and A.C.T.H., Dr Duthie said that the high hopes at first entertained for these substances had not been realised. Though dramatic immediate effects were often obtained, it was clear that an effective cure for rheumatoid arthritis had not been found. Prolonged administration of cortisone and A.C.T.H. was followed by undesirable side effects in about half the patients treated; yet if the drug was discontinued or reduced relapse occurred. However, in some patients—usually those with mild disease—symptoms could be controlled by long-term administration of the drug in small doses. Research was continuing into the effectiveness of new products—notably substance F—and into the use of cortisone and A.C.T.H. in combination with other drugs such as paraminobenzoic acid. But real progress must await a better understanding of the fundamental cause of the disease.

Physiological control: the principle and its application—*Mr Norman Capener* (Exeter) emphasised the importance of affording physiological rest—or physiological control, as he preferred to call it—to a joint affected by inflammation, whether it be the result of rheumatoid arthritis, pyogenic infection or other cause. By physiological control he meant rest with the appropriate amount of function applied physiologically. It was rest with function, and not complete immobilisation. He emphasised that the need for physiological control had not been eliminated by the introduction of antibiotic drugs, cortisone and A.C.T.H. Only by ensuring physiological rest could the resources of the body be directed fully towards the restorative processes.

To put the principle of physiological control into practice he had developed the idea of "liveliness" in splint construction. Instead of providing rigid fixation, splints constructed on this principle allowed a limited amount of controlled movement, yet held the part in the optimum

position between the movements. The chief mechanical force in his "lively" splints was spring wire, used whenever possible as an integral part of the construction or framework of the splint. A range of splints employing the principle of "liveliness" was exhibited.

Minor orthopaedic measures used in conjunction with medical treatment—*Mr John Bastow* (Bath) described orthopaedic adjuncts to medical treatment, which fell into four groups. *Splints*—These were useful as a protection for inflamed joints in the active stage of the disease, and helped to prevent deformity. Care should be taken to maintain affected joints in the optimum position. Shells were preferable to complete plasters. *Palliative operative procedures*—These included procaine nerve block, neurectomy, intra-articular injections, arthrotomy and lavage, and bone drilling. Procaine nerve block was often useful, but neurectomy was disappointing because it seemed impossible by a simple operation completely to denervate a joint. Intra-articular injections were not of value in active rheumatoid arthritis, but they might be in the secondary osteoarthritic stage. Arthrotomy and lavage was a useful procedure in cases of persistent effusion; fibrinous masses were removed and the joint was rested in plaster for a few weeks after the operation. *Bone drilling*—This was at one time used extensively; but it should be confined to cases in which secondary osteoarthritis was associated with extensive sclerosis beneath the joint surfaces. Drilling of the dense bone might often relieve the nocturnal pain which was a common feature of such cases. *Joint manipulation*—This had its uses both in the active stage and in the chronic stage of the disease. In the active stage it should be used only to overcome existing deformity, and should be followed by splintage in the correct position. In the chronic stage manipulation might be used also to improve the range of movement in selected cases. It should be followed by active exercises designed to improve power by progressive increase of resistance. The Guthrie Smith sling exercise apparatus had proved very useful.

Surgery in the treatment of rheumatoid arthritis—*Mr Alexander Law* (London) said that it was often difficult to decide at which stage of the disease orthopaedic operations might prove most useful. He believed, with Smith-Petersen, that surgery had often been left too late and that in certain groups it could be advised at a relatively early stage—that was, when medical treatment was beginning to fail. Operations that might be done early included excision of the acromion for disabling subacromial bursitis; excision of the head of the radius in cases in which spasm of the biceps pulled the diseased radial head up against the humerus; and synovectomy in cases of recurrent effusions with extensive synovial disease in the knee joint. A second group of operations was applicable only when the active stage of the disease had passed. In the elbow, excision-arthroplasty had a definite place. For arthritis of the wrist with ulnar deviation of the hand he advised excision of the lower end of the ulna with arthrodesis of the radio-carpal joint; this gave a much improved position of grasp. For old disease of the knee with extensive destruction of cartilage arthrodesis was still the best treatment. Attempts to form a useful and painless arthroplasty at the knee had so far met with little success. Severe flexion deformity of the spine could be corrected by "osteotomy" in the lumbar region, whether the deformity resulted from rheumatoid arthritis or ankylosing spondylitis. In the hips, mould arthroplasty as developed by Smith-Petersen gave encouraging results although movement and function were both restored less completely than was possible in osteoarthritis. For severe affection of the forefoot amputation of the toes was of established value.

Discussion—*Mr W. D. Coltart* (London) emphasised the enormous difficulties that attended the treatment of rheumatoid arthritis. In some respects they were greater than those of tuberculosis or poliomyelitis. Rheumatoid arthritis often went on for as long as twenty years and this created serious social and economic problems in addition to the clinical problems. He felt that so far as the surgery of rheumatoid arthritis was concerned there were only three operations of established merit, namely arthrodesis of the knee, arthroplasty of the elbow and amputation of the toes; in appropriate cases these were of great value. Speaking of training and research, he said that rheumatologists should have an adequate training in orthopaedics, and orthopaedic surgeons should be prepared to help in the training of rheumatologists. *Mr F. C. Durbin* (Exeter) said that in his experience, and in that of his colleagues at Exeter, synovectomy in selected cases had given a high proportion of gratifying results.

OTHER PAPERS

Complications of Osgood-Schlatter's disease—*Mr R. I. Stirling* (Edinburgh) recalled that though there had been controversy over the cause of Osgood-Schlatter's disease most authorities favoured a traumatic origin. It was well known that in most cases the symptoms settled down after appropriate protection for the knee by plaster, etc.; but that in a few, more severe symptoms

develop for which drilling or bone-pegging operations had been advocated. Complications had been regarded as few, but separation of a bony fragment which remained discrete behind the patella tendon was recognised. He had encountered two further complications which might have important effects. The first was premature fusion of the anterior part of the growing epiphysis at the upper end of the tibia. Continued growth of the posterior part of the epiphysis then led to genu recurvatum, which might be severe enough to require correction by osteotomy. The second complication that he had encountered was upriding of the patella. The patella might be three-quarters of an inch higher on the affected side than on the normal side. This led to undue pressure on the lower facet of the patella and to consequent osteoarthritis. He had treated this complication by transplanting the patellar insertion lower down the tibia; when necessary, this should be done early to prevent the development of arthritis. Describing the technique of the operation, Mr Stirling advised transplanting the tendon insertion with only a thin sliver of bone, which was sutured beneath a prepared flap of periosteum. If a large block of bone was displaced with the tendon the blood supply of the epiphysial region might be jeopardised and anterior epiphysial growth might be impaired.

Discussing this paper, *Mr S. A. S. Malkin* (Nottingham) said that he had had a patient in whom genu recurvatum had developed from disturbance of growth at the anterior part of the upper tibial epiphysis. In his case the condition was without symptoms.

Leg lengthening—*Mr W. V. Anderson* (Edinburgh) described his experiences with leg lengthening operations in seventy-one children. In fifty-six cases lengthening was undertaken to correct deficient growth after poliomyelitis; the remainder comprised a miscellaneous group. The average lengthening gained was 2 inches (shortest gain 1 inch, largest 3½ inches) and the average time of union was five months. The method used was first to lengthen the tendo calcaneus, then to divide the tibia by a long oblique or stepped osteotomy, to fix the fragments on an Abbot type distraction frame by double fixed pins above and below, and to lengthen the limb by screw distraction at the rate of ¼ inch per day. When union was moderately advanced the limb, with the pins, was transferred to a walking plaster. Complications were few. There had been no vascular catastrophes, and no anxiety on this score. Minor pin-track sepsis was common; in one case severe infection made it necessary to discontinue the lengthening. Another patient developed a chronic bone abscess. Angulation of the fragments, with consequent pressure on the skin, should be corrected early if it arose. A pin had broken in one case. The most serious complication was the development of valgus deformity of the foot. Failure of the lateral malleolus to come down with the tibia was largely responsible. He had overcome this in recent cases by fixing the lateral malleolus to the tibia by a pin or screw at the time of operation; this had sometimes been combined with division of the peroneal tendons. In his experience the formation of callus was not inhibited by the lengthening process, at any rate in children.

Congenital arteriovenous fistulae of the extremities—*Mr Douglas Robertson* (Edinburgh) had studied arteriovenous fistulae of the extremities at the vascular units of St Bartholomew's Hospital, London, and of the Edinburgh Royal Infirmary. He recalled that during development the limb circulatory system consisted initially of a network of capillaries. Normally, many of these disappeared, while others developed to become major vessels. Persistence of the primitive network might lead to arteriovenous fistula. The effects of arteriovenous fistulae were local, regional and systemic. Locally, there were dilation and arterialisation of veins. Regionally, there was hypertrophy of the limb (with increase of length if the epiphyses were open) with increased skin temperature. Systemically, there was diminished peripheral resistance, with normal systolic and lowered diastolic blood pressure. There might be increase of blood volume, enlargement and dilation of the heart, and a "bradycardiac reaction" (slowing of pulse rate on compression of the main artery of the affected limb). In the investigation of these cases a general examination with careful assessment of the cardiac function was essential; special examinations included radiography of the limb and heart, and oxygen saturation tests (increased saturation above 60 per cent was not, however, diagnostic of arteriovenous fistula, as had been thought). Clinically arteriovenous fistulae fell into two types, local and diffuse. In the *local* group, the characteristic signs—venous and arterial dilation, raised skin temperature, and local bruit—were present. Though localised, the abnormal arteriovenous connections might be marked. Arteriography was useful in showing their extent. As to prognosis, the tendency was for more and more blood to be short-circuited as time went on. Ulceration and gangrene were common ultimate complications. Ideally, treatment should be by local excision which, however, was seldom possible. Ligation of proximal arteries was ineffective and might even make matters worse. Amputation might become necessary.

The diffuse type of lesion was the type that might lead to "giant limb." The earliest

abnormality was increase of length and girth of the limb. Usually there were surface haemangiomatous lesions, increased surface temperature and dilation of vessels. A form of streptococcal cellulitis was a recognised complication: another was recurrent haemarthrosis of the knee joint. In the diffuse type arteriography was of little value. As to prognosis, the appearance of the limb was a guide to the extent of the abnormality. If lengthening alone was the main feature the prognosis was relatively good. But if vascular effects were predominant the prognosis for the limb was poor. If it seemed that the limb could be preserved, leg equalisation programmes could be considered.

Aortic obstruction—*Professor Sir James Learmonth* (Edinburgh) described the features of aortic obstruction from embolism and thrombosis, with consequent ischaemia of both lower limbs. He emphasised the importance of the visceral vessels, especially the marginal artery of the colon, in the collateral circulation. *Acute aortic block* was caused by embolism or pseudoembolism (spasm superimposed upon small branch embolism). True emboli usually arose in the heart, and most patients with acute aortic obstruction had cardiac disease. Occasionally a large embolus might be set free on the venous side of the heart and pass through a large interatrial septum. Consecutive thrombosis was to be feared in cases of acute block in which diagnosis and embolectomy were delayed. Clinically, acute obstruction was characterised by sudden onset of limb pain; ischaemic paralysis developed and after six hours the changes became irreversible. After early embolectomy recovery proceeded centrifugally. Treatment was by heparin injection and embolectomy. Further episodes were to be feared; they might be prevented by excision of the left auricle. *Chronic aortic block*, due to consecutive thrombosis, might exist in severe degree with remarkably few clinical features. Usual findings were lower limb wasting; some analgesia; swelling with posture changes; and impotence. There might be localised gangrene, or claudication at short distances. Patients had survived for seven to ten years. In the investigation of these cases arteriography was disappointing. Blood-flow tests showed fairly good flow, but at low pressure. In treatment, a conservative régime was the wisest. Sympathectomy was disappointing, and excision of the affected segment was not advised.

Giant-cell tumours—*Professor Robert McWhirter* (Edinburgh) discussed the possible relationship between giant-cell tumours, osteitis fibrosa and simple bone cysts. In considering such a relationship he emphasised the importance of taking all the evidence into account—clinical as well as radiological and histological. Clinically, he noted particularly the location of giant-cell tumours in vascular regions such as the upper end of the humerus and the growing ends of the tibia and fibula. Bone cysts, on the other hand, were found in relatively avascular areas such as the lower end of the humerus. Cysts occurred in children, whereas giant-cell tumours were commonest in the third decade. He had noted also that lesions developing at the growth cartilage, such as dyschondroplasia, could be found throughout the shaft of the humerus, the location depending upon the age at which the defect arises. Correlating all the evidence, he submitted that simple bone cysts, localised osteitis fibrosa and giant-cell tumour arose from one and the same defect of the growth cartilage, the defect existing for a short period only and being followed by normal growth. The initial defect consisted simply in the laying down of fibrous tissue instead of bone. If the defect arose at an early age—for example at the upper end of the humerus—it progressed distally in the course of growth and reached an area of relatively poor blood supply; the fibrous tissue consequently degenerated to from a cyst. If the defect arose near the end of the period of growth, it would not migrate distally and would remain in an area of rich blood supply, which would enable the lesion to expand and become a giant-cell tumour. That was why giant-cell tumours were found in the region of vascular epiphyses. Local osteitis fibrosa cystica was intermediate between simple cysts and giant-cell tumours. The lesion did not degenerate to form a cyst, but its blood supply was insufficient to allow development into giant-cell tumour. If these three lesions were part of the same process, a generic term to denote all three would be "dysfibroplasia." If his view was correct, it was clear that giant-cell "tumours" were not true tumours any more than were the localised cystic lesions of hyperparathyroidism.

Discussing this paper, *Mr Roland Barnes* (Glasgow) said that although the three lesions mentioned had points of similarity, they were also dissimilar in many respects. Some giant-cell tumours grew so rapidly that they could not possibly be accepted as dystrophic lesions; others gave rise to metastases. These considerations made him critical of the hypothesis. *Dr J. F. Brailsford* (Birmingham) recalled that malignant metaplasia could occur in dysplastic lesions. He showed radiographs of a lesion diagnosed radiographically as giant-cell tumour which, though no treatment had been given, had nevertheless healed. *Professor McWhirter* (closing) said that histological findings were often confusing. Malignant change could arise in any tissue, but malignant giant-cell tumour must be rare.

Naviculo-cuneiform fusion in the treatment of flat foot—*Mr E. A. Jack* (Edinburgh) felt that severe flat foot ("unstable everted foot") was so disabling as to merit correction whenever possible. He believed that the long arch depended upon the integrity of joints and ligaments, and that muscles could not maintain an arch in which there was a ligamentous defect. Studies of lateral radiographs showed that in a normal foot the axes of the first metatarsal, cuneiform, navicular and talus were in a straight line, which formed an arch with the calcaneum. In flat foot there was a sag in this line. Sag occurred either at the talo-navicular joint alone, at the naviculo-cuneiform joint alone, or at both these joints; naviculo-cuneiform sag being commoner than talo-navicular sag. The great-toe raising test was useful in diagnosis; raising of the great toe corrected the dropping of the arch if the sag was in front of the navicular. On the basis of these observations he had treated forty-six feet by naviculo-cuneiform fusion in the corrected position, with satisfactory correction in thirty-eight feet. The operation was applicable only to those cases in which the dropping of the arch was shown to be due to naviculo-cuneiform sag.

Femoral head changes in congenital dislocation of the hip—*Mr G. W. Baker* (Edinburgh) had reviewed 120 cases of congenital dislocation of the hip treated at the Margaret Rose Hospital, with special reference to the condition of the femoral head. Many femoral heads showed changes in texture but without change of shape. In twenty-six cases there were well marked changes in outline of the femoral head, appearing first as a defect at its lateral margin. In twenty-one, healing occurred with restoration of good shape; in five, gross deformity remained. The appearance did not suggest a process like that of Perthes' disease. There were no metaphysial changes, no fragmentation and no anterior defect such as were seen in Perthes' disease. He had tried to correlate the femoral head changes with the original appearance and methods of treatment. The evidence did not suggest that the trauma attending manipulative reduction was the cause. The cause might be found in direct local pressure against the femoral head while in the position of immobilisation. In an attempt to prevent femoral head changes, he was now treating congenital dislocation of the hip by a period of preliminary traction; then, after reduction, immobilisation on a frame with continuous traction.

Diastasis of the tibio-fibular syndesmosis—*Mr J. G. Bonnin* (London) said that diastasis of the tibio-fibular syndesmosis could be classified into three degrees. First degree diastasis occurred when there was rupture of the anterior tibio-fibular ligament, with a helical fracture of the fibula at the lower third. The force responsible was a lateral rotation force. Second degree diastasis followed rupture of the anterior tibio-fibular ligament and of the interosseous ligament. It was associated with high fracture of the fibula. In third degree diastasis there was rupture of the anterior tibio-fibular, interosseous, and posterior tibio-fibular ligaments, and fracture of the fibula. Second and third degree types were caused by a combination of lateral rotation force and abduction force.

Discussing the significance of diastasis, Mr Bonnin said that it demanded a relatively long period of immobilisation as compared with fractures without diastasis. He considered that operative fixation was seldom necessary, especially if all three ligaments were not ruptured. In most cases, provided the medial elements of the joint were well reduced (if necessary by screw fixation of the medial malleolus) stable reduction of the diastasis could be maintained.

Instability of the ankle joint—*Mr A. C. Bingold* (Sidcup) had studied sixty-six patients with instability of the ankle (fourteen bilateral). The cases fell into two groups. In the first group instability resulted from tears of the lateral ligament (five cases). The second group comprised a variety of conditions the common factor among which was a tendency to walk on the outer border of the foot; the commonest were hallux rigidus (forty-one cases) and pes cavus (twenty cases). Treatment of the first group was by reconstruction of the lateral ligament when the tear was complete, or by appliances if the tear was incomplete. In the second group treatment was usually by outside heel floats, detorsion supports, or concealed springs, with operative correction of the underlying defect when necessary.

Rotational lordosis of the spine—*Mr E. W. Somerville* (Oxford) reported his preliminary studies of scoliosis based on a "very old idea," that of William Adams to the effect that rotation plus lordosis equals scoliosis. Theoretically, scoliosis could arise from failure of the posterior elements of the spine to grow while the anterior elements continued to grow. The first effect would be lordosis, which could be corrected by rotation of the spine in the area of the deformity, followed by lateral flexion. Mr Somerville illustrated this concept by an ingenious model which showed that attempts to correct a lordotic curve led easily to rotation, especially under the added influence of gravity. If this mechanism was correct, he continued, interference with the growth of the posterior elements of the spine should lead first to lordosis and then to scoliosis; and as the curve

increases so should the rotation. This had in fact happened in a rabbit, the posterior elements of whose spine had been restrained mechanically at operation. The scoliosis that had resulted approximated more closely to the typical human rotato-scoliosis than did that produced experimentally in animals by restraining growth on one side of the spine. In short, the hypothesis on which he was working was that rotato-scoliosis might be nothing more than the result of failure of growth of the posterior elements of the spine.

Orthopaedic aspects of vocational training of the severely disabled—*Mr E. Stanley Evans* (Alton) recalled that Robert Jones's idea of "curative workshops," in which training was begun during treatment, had fallen into abeyance after the first world war. But a long course of vocational training *after* the conclusion of a long period of institutional treatment was more than most adult patients were prepared to face; and recently schemes of relatively short training, commenced during treatment, had been introduced. Such courses were sufficient to fit men for industry, without turning out skilled craftsmen such as did the longer courses of training. Each disablement problem should be considered individually, and with due regard to the education, mentality and physical ability of the patient. Whenever possible, patients should be trained for jobs that they wanted to do. Physically, it was necessary to consider what a patient could do, rather than what he could not do. Disabilities of the lower limb presented relatively few difficulties; disabilities of the upper limb often presented difficulties, and in many cases personal help (in dressing, toilet, etc.) was as necessary as training for a job—especially in cases of double hand amputations. Among the trades for which these men had been found suitable were draughtsmanship, gardening, welding, painting and plastering. The co-operation of large industrial firms was important—as shown by the great work done by Austin Motors and Vauxhall Motors—and opportunity should be available during the training stage for periodical orthopaedic review and, if necessary, appropriate treatment.

Arthroplasty of the hip—*Dr Robert Judet* (Paris) described his experience of "resection-reconstruction" of the hip, which he had developed in conjunction with his brother during the recent war. The method was a logical development of resection of the head of the femur (Whitman), and his acrylic prosthesis now used was evolved after many trials. Describing his operative technique, he said that he preferred the anterior incision, which caused little anatomical disturbance. The prosthetic head should be a close fit in the acetabulum—if it was too loose a fit weight was taken on only a small area and pain was to be feared. The acetabulum should be made deep enough to contain the whole head. After operation, lateral rotation should be prevented as a safeguard against dislocation. He had experienced few complications in the 400 cases of his series. The mortality rate was 1·75 per cent and infection rate 3·8 per cent—a figure that he felt was too high. Breakage of the stem had occurred before the present pattern with steel reinforcement had been evolved, but not since. Dislocation had occurred fourteen times; it was specially prone to occur in cases of old congenital dislocation of the hip, and in old neck fractures with short stump. Nevertheless there had been no further dislocation since adequate steps had been taken to prevent lateral rotation. Of his 400 operations, 216 were done for osteoarthritis, forty-seven for non-union of cervical fractures and seventy-seven for old congenital dislocation of the hip in adults. Results had been assessed on a "points" basis, taking into account relief of pain, movement and gait. In osteoarthritis 64 per cent were good or excellent, and 36 per cent were moderate or bad. In ununited fractures the results were universally good. In old congenital dislocation results were good in about one-third of all cases, moderate in a third and disappointing in a third. Speaking more specifically of the operation as done for old congenital dislocation, Dr Judet said that he did not favour trying to bring the head down to the level of the original acetabulum. It should be brought down slightly—about 3 or 4 centimetres was the best—to improve the function of the abductor muscles. The new socket should be deepened if necessary to the floor of the ilium or even through it. The operation should be done only for pain, never for limping alone.

The development of osteoarthritis of the hip joint—*Mr K. H. Pridie* (Bristol) had studied in detail the structure of femoral heads removed at operation in cases of osteoarthritis, using, among other methods, the technique of microradiography of thin sections. He believed that the primary factor in every case was a vascualr "catastrophe" in the head of the femur or in the acetabulum, whether or not there was a predisposing cause such as disproportion (as in old congenital dislocation, old Perthes' disease, etc.), or a vascular accident (as after injury, or in Caisson disease). Examination of many femoral heads had shown cysts or lesions below the surface. Over these areas the cartilage, deprived of its blood supply, was worn away.

The treatment of Pott's disease by curettage of the spinal lesion—*Mr M. C. Wilkinson* (Braintree) reported preliminary clinical trials of the treatment of selected cases of tuberculosis of the thoracic spine by removal of pus and granulomatous tissue adjacent to the lesion. Eight children

and fifteen adults had been operated upon. All had radiographic evidence of local abscess or granuloma, but no paraplegia (the treatment of Pott's paraplegia was not considered in this paper). The object was to remove all pus and as much granulomatous tissue as possible, on the supposition that persistent abscess or granuloma retarded healing. Before operation the patient's resistance was raised by a preliminary period of general treatment; and streptomycin was administered before and after operation. The evacuation was carried out through a modified costo-transversectomy approach through which access to the vertebral bodies was obtained; necrotic material was curretted away from the affected vertebral bodies after evacuation of pus. The wound was closed by suture. Results were promising in twenty of the twenty-three cases; in a favourable case serial radiographs showed no further destruction of bone after the operation, and the affected bones showed progressive evidence of healing.

"Osteogenesis exhausta"—*Dr J. F. Brailsford* (Birmingham) described the clinical and radiographic features of an unusual generalised affection of bone. The patient, now aged sixty-three, had had back pain and disabilities of the lower limbs for twenty years. Radiographs showed a widespread generalised skeletal affection, in which there were multiple zones of decalcification (Looser's zones) in many bones, especially the pelvis, and multiple "Milkman's" fractures, notably of the metacarpals; yet there was no bending of the bones, and the skull was unaffected. Biochemical investigations showed no abnormality. Dr Brailsford was of the opinion that the condition was of long standing and represented an exhaustion of osteogenesis. He emphasised that so-called Milkman's fractures could occur in a number of different affections and did not constitute a distinct disease.

Films—Films were shown on arthroplasty of the hip by *Mr H. Osmond-Clarke* and *Mr W. A. Law*, and on the treatment of non-union of the carpal scaphoid by excision of the radial styloid and bone grafting by *Mr D. F. Thomas*.

CLINICAL CASES

The clinical part of the meeting was held during the afternoon of October 26 in the Royal Infirmary. From the array of interesting clincial material several groups of cases were demonstrated.

Popliteal nerve cyst—*Mr A. J. Slessor* described three cases of popliteal nerve cyst, and showed another a year after excision of the affected segment. The cyst had developed in the lateral popliteal nerve, and its removal had necessitated complete excision of the involved segment of nerve; suture of the cut ends had led to full recovery of function.

Acrylic replacement of upper part of femur for osteoclastoma—*Professor Walter Mercer* demonstrated a young man in whom, ten months previously, he had resected the upper five inches of the femur, the seat of a benign giant-cell tumour which had failed to respond to radiotherapy, and in which a pathological fracture had occurred and failed to unite. The resected portion was replaced by an acrylic prosthesis based upon the Judet acrylic head, secured to the shaft of the femur by two long vitallium plates. Function was good, and there had so far been no sign of recurrence.

Streptomycin and synovectomy in tuberculosis of the knee—*Professor Walter Mercer* also showed a young girl in whom he had performed a synovectomy of the knee for tuberculous synovitis. The operation had been preceded and followed by systemic streptomycin, and the excellent present condition—normal range of movement, no effusion, and strong quadriceps—was apparent.

Treatment of cerebral palsy—*Mr G. A. Pollock* explained the Temple Fay method of treating cerebral palsy. This method, followed at the Edinburgh School for Spastic Children, aimed at the development and organisation of the automatic spinal reflexes as observed by Sherrington and Babinski in the decerebrate animal, and the ultimate co-ordination of these reflexes with what remained of the higher cortical control. As a first step, the cerebral palsied patient was considered as an amphibian—that is one who, because of partial or complete loss of cortical control, must depend for function upon the mid-brain. In practice it was found to be far easier to teach the simple movements excited at this level than to try to instruct in more complicated movements needing cortical control. The child was placed prone with chin forwards on a polished floor previously dusted with french chalk, or on a blanket covered with american cloth. Aided by a physiotherapist, he was made to flex the arm and leg of one side, the extended thumb pointing towards the face, which turned towards the hand. As these limbs swept out and down again, those of the opposite side would flex in turn to a similar degree, the head rotating towards the flexed arm. The child kept up a regular rhythm of alternating movement aided by music until he

developed a definite pattern. The original uncertain jerky movements became replaced by a smooth series of regular muscle contractions. Once the homolateral or amphibian pattern was well developed the child progressed to the reptilian or next stage of evolution, characterised by the crossed or contralateral pattern of movements. As the right arm rose, the left hip would flex and the head turn towards the advancing hand. After two to three months of instruction, children were usually capable of carrying out these crossed movements regularly by themselves without further assistance from the physiotherapist. Many of them developed a remarkably easy flow of rhythmic co-ordinated muscle action. Once the crossed pattern was well established further advance was by progressive patterns of movement through the stages of creeping and crawling to independent walking, feeding and writing.

Aortic obstruction—*Miss Catherine Burt*, in illustration of Sir James Learmonth's paper, showed three patients in whom obstruction of the aorta had occurred. The first, a woman of fifty-seven, had developed a pulmonary embolism during the treatment of varicose veins. During recovery from this the circulation to both lower limbs was suddenly cut off by a large embolus which blocked the aorta at its bifurcation. There was complete loss of power, sensation and pulses in both limbs, but full recovery followed removal of the embolus, through the common iliac artery, within three hours of impaction. A miner aged forty-six, already suffering from mitral stenosis and auricular fibrillation, had suddenly lost the power of both lower limbs and also sensation and palpable pulses below the mid-aorta. Five hours later an embolus was removed from the aorta via the common iliac artery; rapid and practically complete recovery occurred. Five weeks after the embolectomy the auricular appendage was removed by Mr Logan; it contained remnants of old adherent clot. Now, with full function in both lower limbs, though still with fibrillation, the patient had returned to his previous work in the pit. The third patient, a man of about sixty, illustrated aortic obstruction from thrombosis, due to arteriosclerosis and believed to be slowly progressive. He was severely handicapped by intermittent claudication, and though no improvement in this could be expected, bilateral lumbar sympathectomy was performed to increase the circulation to the skin of the feet, and in this it succeeded.

Massive grafts in inveterate non-union of forearm and tibial fractures—*Mr I. L. Dick* described a case of long-standing non-union of both bones of the forearm that had resisted previous bone-grafting operations. Double onlay cortical grafts with iliac chips had resulted in sound union and remarkably good function. He also illustrated a case of a girl with ununited fracture of the upper third of the tibia, who had been too young when first seen to provide autogenous bone; a graft from her sister proved unsuccessful. Further operation was postponed until the child was bigger, and then a cortical graft with iliac chips brought about sound union.

Limb equalisation—*Mr R. I. Stirling* demonstrated a girl in whom tuberculous disease of the hip had led to six inches of shortening of one lower limb. Equal length was achieved by lengthening the femur and tibia on the affected side and shortening the femur on the sound side. He showed an apparatus whereby damage to the knee joint during femoral lengthening was prevented by transmission of pressure past the joint instead of through it.

Treatment of congential pseudarthrosis of tibia and fibula—*Mr A. M. Stewart* showed a small child of eighteen months, born with pseudarthrosis of each tibia and fibula. Constant immobilisation in plaster casts, and subsequently perspex splints, with prevention from all weight-bearing, had brought about union in excellent line of all the bones except one fibula. The child had been taught to walk in patten-ended calipers, but union was now so sound that these could probably be discarded.

Supracondylar fractures with circulatory impairment—*Mr J. J. Mason Brown* described six cases of supracondylar fracture with circulatory complications. On admission, all had shown impairment of the peripheral circulation—absence of the peripheral pulses, coldness of the limb and impaired capillary circulation with rubor or cyanosis. Delay in admission to hospital, varying from four to twenty-eight hours, was an important factor. Impairment of circulation must take priority in treatment but, of course, reduction of the fracture might restore the circulation. In these six cases the artery was explored immediately, or after manipulation of the fracture when this was not followed by improvement in the circulation. In two cases the artery was kinked between the fragments; in two it was attached to a fragment and kinked by the fascial band; and in the remaining two no cause was found for the severe arterial spasm. Swelling and tension in the cubital fossa made it advisable to leave the wounds open and they were closed later (average—four days) by delayed suture. In one case, the upper forearm was so tense that an incision was made through the skin and deep fascia allowing the muscle to prolapse through it;

this incision was closed by delayed suture some days later. The anterior incision allowed reduction of the fracture under vision. In no instance was there evidence of damage to the vessel wall, and the artery was left alone but for the application of warm saline and freeing where necessary. After the exploration reflex vasodilatation and alcohol (an excellent vasodilator in young children) were employed. In all six cases there was an excellent functional recovery of the elbow, forearm and hand; and Volkmann's contracture did not ensue. He considered that exploration could do no harm and that it might prevent damage to the artery by manipulation of the fragments in those cases in which it is caught between them. Possibly there would have been no ischaemic changes in any of these children if treated by manipulation of the fracture alone, but relief of tension is an important help in maintaining an adequate circulation during the critical days.

ANNUAL GOLF COMPETITION OF THE BRITISH ORTHOPAEDIC ASSOCIATION

The Annual Golf Competition of the British Orthopaedic Association was held at Cambridge on April 6, 1951. Fine weather graced the occasion and the Mildenhall Golf Course was in excellent condition. Twenty players turned out, and an exciting finish resulted in a tie between J. V. Todd (Newcastle-upon-Tyne) and N. W. Roberts (Liverpool), with scores of 75 net. After some free discussion it was decided that the lateness of the hour precluded a play-off and that they should be considered joint holders of the Cup for 1951. An informal dinner to the competitors was held in the evening.

It is perhaps not widely known that the Robert Jones Cup was presented by the late Mr Rowley Bristow to promote "sport and good fellowship" among the members of the British Orthopaedic Association. The fact that Mr Bristow presented the cup was not hitherto engraved on it, but this omission has now been rectified.

Through the generosity of an anonymous donor in the Association, the cup now rests in a very handsome red leather case, which shows off the graceful lines of the cup to the best possible effect. The thanks of all golfing members of the Association are due to the generous donor of this splendid gift.

UNIVERSITY OF LONDON

INSTITUTE OF ORTHOPAEDICS

A study of 762 patients with spinal cord injuries—*Dr Loyal Davis*, Professor of Surgery, Northwestern University, Chicago, accepted an invitation to attend as guest lecturer a Clinical Conference on October 11, 1951, and he gave an account of experience in the care of 762 patients with spinal cord injuries treated at a special centre in Chicago. This centre is one of four permanent veteran hospitals in the United States of America, which are under the control and medical care of civilian teaching hospitals and between them look after 2,000 paraplegic patients. These patients had received an enormous amount of public attention, far greater than any other war disabled. Although a vast array of specialists was required to deal with the manifold problems of the paraplegic—under the co-ordinating responsibility of the Department of Neurology—it had been found essential for each patient to have his own regular medical attendant, acting in the capacity of a personal physician and adviser. Each ward of twenty-five patients came under the care of a young neurologist assisted by a neurosurgical trainee.

Dr Loyal Davis gave clinical data of the problems encountered. The only indication for surgical intervention in traumatic paraplegia was the presence of a demonstrable block in the subarachnoid space. As a general guide, it should be taught that no fracture or fracture-dislocation of the spine with cord damage should be operated upon unless there is a demonstrable subarachnoid block, and this was true even for a fresh fracture-dislocation.

Infections of all kinds, and particularly of the genito-urinary tract, still constituted the most serious danger to life. An indwelling catheter, periodically released until tidal drainage could be instituted, was the best method of preventing infection and promoting automatic micturition. Enthusiastic nursing had almost eliminated bedsores and much of the work of the plastic surgeon.

A detailed study of micturition in 471 patients had been carried out. This showed that with proper management, automatic micturition would always develop after complete lesions. It developed earliest and was most efficient in lesions between C.7 and T.5. There was a large amount of residual urine. Cystograms might indicate when a trial of automatic micturition should be started. Reflex phenomena, sepsis or general ill-health might severely hamper or prevent efficient automatic micturition.

Pain, which was commoner in military than in civilian cases, was of three types: 1) root pain; 2) visceral pain; and 3) localised burning and tingling pain, much influenced by emotion or

environment; this important and very common pain does not conform to a dermatome, and the origin is obscure. Dr Davis's experience controverted the previous evidence that somatic stimuli below the level of a complete lesion could produce pain via sympathetic fibres entering the cord above the level of the lesion.

The distressing pathological reflex responses other than those from bladder or bowel stimuli were so variable that no rules for their occurrence could be laid down. They were aggravated by sepsis, especially bladder sepsis; patients ideally cared for did not have severe spasms. The severity was related also to the level of the lesion: the higher the lesion the worse were the spasms. Seemingly they were uninfluenced by sedatives. In properly selected cases anterior rhizotomy was uniformly successful if carried out over enough segments. Neither posterior rhizotomy nor resection of the ends of the divided cord had proved helpful. Adductor spasm could be relieved immediately by bilateral retroperitoneal obturator neurectomy. Tenotomies also had been carried out.

Interesting and occasionally distressing disturbance of sweat-regulation had been encountered and studied. Many patients suffered from diffuse general sweating although their lesions were as high as T.4. When the preganglionic sympathetic outflow was damaged, normal thermo-regulatory sweating did not occur, but postganglionic sweating in response to various drugs could still be produced. The spinal reflex sweating that occurred below the level of a complete lesion was ascribed to the survival of the sympathetic system for as long as even ten years.

Reflex defaecation had been attained in 131 out of 144 patients, even in those with complete lesions of the cauda equina. Enemata should be dispensed with after a few days and the patients instructed in the initiation of reflex defaecation by stimulation of the rectal wall.

Some general metabolic observations had been made. Basic blood pressure in cervical lesions was consistently low and did not show the normal increase with age. Urinary concentration was defective and the D/N ratio altered, although the kidneys were normal. The glucose tolerance test showed abnormalities although the liver function was unimpaired. There was considerable loss of weight; digestive function was disturbed; and the intake of water was greatly increased.

The return of sensibility usually occurred from above downwards but was occasionally patchy or might even begin distally. The return took place according to the anatomical pattern of tracts in the spinal cord, and not according to the type of sensibility. Motor return was less frequent than sensory return, and return of sphincter function was less frequent than other motor functions. The longest interval between injury and the first return of function was four years. The quality of recovery was not related to the duration of paralysis. A fair number of patients had been made socially and economically independent within the limits of their disability.

UNIVERSITY OF LIVERPOOL

DEGREE OF MASTER OF ORTHOPAEDIC SURGERY

The successful candidates in the December 1951 examination for the degree of Master of Orthopaedic Surgery of the University of Liverpool were:

W. J. Betts, Adelaide, Australia;
P. Chandra, Jamangina Bad, India;
K. R. Daymond, Sydney, Australia;
G. F. Fischer, Bloemfontein, South Africa;
F. J. Gruar, Hamilton, New Zealand;
D. R. Gunn, Liverpool, England;
A. K. Gupta, Patna, India;
M. Natarajan, Madras, India;
J. A. O'Garra, Liverpool, England;
J. S. Roarty, Killara, Australia;
I. W. Winchester, Edinburgh, Scotland;
W. H. de Wolfe de Wytt, Liverpool, England.

The Directors' Prize was awarded to F. J. Gruar of New Zealand. The external examiners were Sir Reginald Watson-Jones and Professor Walter Mercer.

ROYAL COLLEGE OF SURGEONS OF EDINBURGH

Election of Professor Walter Mercer as President of the Royal College of Surgeons of Edinburgh—Three years ago we recorded that Mr Walter Mercer had been appointed Professor of Orthopaedic Surgery in the University of Edinburgh (Journal of Bone and Joint Surgery, **31-B**, 142). We now learn with much pleasure that this brilliant and versatile surgeon has been elected President of the Royal College of Surgeons of Edinburgh.

REGIONAL ORTHOPAEDIC CLUBS

EAST ANGLIAN ORTHOPAEDIC CLUB

A meeting of the East Anglian Orthopaedic Club was held at the Thorpe Hall Rehabilitation Centre, Peterborough, on June 23, 1951. Clinical cases were demonstrated.

Polyostotic fibrous dysplasia—*Mr R. Britt*, introduced by Mr Noel Smith, demonstrated a case in a boy with a pathological fracture of one femur. It was found on radiography that many of his bones were affected, including the sella turcica, which had the club-shaped clinoid processes described by other observers. There were pigmented patches in the skin and the typical facies of a large vault to the skull and a small face. Blood chemistry was normal and no familial factor was apparent.

Congenital infantile coxa vara—*Mr Szarnagiel*, introduced by Mr Noel Smith, showed a family of father and three sons suffering from this condition. When first seen in one boy the condition was thought to be rickets, but when it was noticed that other members of the family were similarly affected and that the blood chemistry was normal, the diagnosis was revised. The films of the boys each showed the triangular fragment of bone on the lower part of the neck of the femur described by Fairbank; the head of the femora were unusually translucent and the line of the epiphysis almost vertical. Two of the boys were examined by those present and it was suggested that they should have subtrochanteric osteotomies carried out in an attempt to correct the coxa vara and rolling gait.

Bilateral cystic medial cartilages in a child—*Mr K. Cleminson*, introduced by Mr Noel Smith, demonstrated a boy of five years of age. His father thought the condition had always been present. On examination a similar smaller cyst was found on the medial side of the left knee. The bilateral nature of the affection supported the view that the cysts were of congenital rather than traumatic or degenerative origin. The radiographs showed widening of the joint space on the medial side of the joint. In discussion those present felt that the cysts should be explored and removed.

Osteoclastoma of upper end of ulna—*Mr Noel Smith* showed this case in which the upper end of the ulna was removed and the space filled with bone chips. After a period of recession there was gradual recurrence. A section of the tumour was then taken for examination, which suggested that the tumour was a secondary deposit from a thyroid carcinoma, though there was no clinical evidence of a primary lesion. The upper end of the ulna was excised and replaced by a tibial graft. The patient returned to work but two years later there was found to be further destruction of the ulna and commencing destruction of the graft. Amputation was carried out and the growth was seen to be of similar structure.

Ruptured diaphragm and multiple injuries—*Mr K. Cleminson* and *Mr Alexander*, introduced by Mr Noel Smith, demonstrated two such cases. The first patient was severely shocked on admission and was given four and a half pints of blood. She was found to have a fracture of the first lumbar vertebra, fracture of ribs, fractured pelvis and acetabulum and a shadow in the chest which was later found to be colon. Three weeks later she developed paraplegia with incontinence of urine and bedsores. She was treated in a plaster bed and later a local spinal fusion was undertaken. A thoracic surgeon carried out decortication of the right lung and repaired the rent in the diaphragm. The second patient was treated as an emergency and the 9-centimetre rent in the diaphragm was sutured.

Excision of calcaneum for fracture—*Mr K. Cleminson* showed a case of comminuted fracture of the calcaneum treated by excision. The man had fallen sixteen feet, landing on his heels on concrete. The calcaneum was excised and the tendo calcaneus sutured to the plantar fascia. Two and a half years had elapsed since the operation and he could now walk several miles and complained of no pain at all. He could stand on tip-toe, in spite of hallux rigidus.

Periosteal sarcoma of femur—*Mr Szarnagiel* showed this patient, a man who had originally reported with a painful swelling of the thigh. Radiographs showed a typical appearance of periosteal sarcoma, which was confirmed by biopsy. Disarticulation at the hip had been carried out, but when he was on the point of leaving hospital, recurrence was noticed in the groin. An extensive dissection of the glands and excision of the scar was carried out, virtually leaving only skin. This was followed by X-ray treatment and the man is alive and well six years after the diagnosis of his condition.

Gross bone loss in fractured femur—*Mr Noel Smith* showed a case in which there was gross bone loss following a road accident. There was spontaneous regeneration of bone; this was reinforced by tibial graft and bone chips. Healing ensued with no shortening and good knee movements.

SOUTH-EAST METROPOLITAN REGION ORTHOPAEDIC CLUB

A meeting of the South-East Metropolitan Region Orthopaedic Club was held at the Brook Hospital, Woolwich, on December 1, 1951. About fifty members and guests were present. A wide variety of cases was shown by Mr E. Lindsay, Mr D. Le Vay, Mr J. Buck and Mr Stack, and certain of these cases were presented for discussion. There were also demonstrations of automatic clinical photography by Mr Brian Stanford, and of specimens of bone tumours by Mr Le Vay.

CANADA

UNIVERSITY OF TORONTO

Professor of Surgery—We are delighted to learn that Dr F. P. Dewar has been appointed Assistant Professor in the Department of Surgery in the University of Toronto, and Surgeon-in-Chief in the Division of Orthopaedic Surgery at the Toronto Service Hospital. Born in Saskatoon, Saskatchewan, in 1911, Dewar graduated from the Toronto Medical School in 1936 with the degree of M.D. and during the years that elapsed before the outbreak of war he filled a number of posts in surgery, anatomy and physiology in Toronto. In 1940 he joined the Royal Canadian Army Medical Corps, in which he held important surgical posts, first in Great Britain and later in Canada. While in Britain he gained the Fellowship of the Royal College of Surgeons of England. After the war he undertook postgraduate work in the Hospital for Special Surgery in New York, being posted as a Fellow in Orthopaedic Surgery; subsequently he passed the examinations of the American Board of Orthopaedic Surgeons. Dewar is already well known to many British orthopaedic surgeons who will join with us in wishing him unbounded success in his new appointment.

Dr F. P. Dewar—Appointed Assistant Professor in the Department of Surgery, University of Toronto.

UNION OF SOUTH AFRICA

SOUTH AFRICAN ORTHOPAEDIC ASSOCIATION

Hitherto, orthopaedic surgeons of the Union of South Africa have been identified as an orthopaedic group of the South African Medical Association. At a meeting held in Johannesburg in October 1951 a South African Orthopaedic Association was constituted. There are thirty-eight foundation members and despite the difficulties of travel over many hundreds of miles, no less than thirty-one attended the meeting at which there was spirited exchange of views and experiences, and a vigorous demand for the creation of a South African Orthopaedic Association. Four orthopaedic surgeons from Southern Rhodesia attended the meeting. It is hoped that this newly formed Orthopaedic Association within the British Commonwealth of Nations will be recognised promptly, that it will be received at the Joint Meeting of Orthopaedic Associations of the English-speaking world in July 1952 in London on parity with the older Orthopaedic Associations of Canada, Australia, New Zealand, Great Britain and the United States, and that the president of the South African Orthopaedic Association will receive from Her Majesty the Queen a jewel of office similar to that which is to be presented to the presidents of the other Orthopaedic Associations. We are delighted to learn that the surgeons of South Africa, who have already made an important contribution, and whose promise for the future development of orthopaedics is so great, have taken the important step of creating an Association by which to increase and enhance their contribution. The new president is Mr G. T. du Toit of Lister Building, Johannesburg, and the secretary, who succeeds Arthur Helfet of Cape Town, is W. T. Ross of Lister Building, Johannesburg. Mr Fouché has been elected emeritus president.

The other members of the Executive Committee for 1952 are Messrs C. T. Moller, G. F. Domisse and C. Morris. The Association will be represented at the South African Medical Association Congress in 1952 by Mr H. Bell as Chairman and Mr H. I. Maister as Vice-Chairman. Mr S. Sacks was appointed external examiner for the Orthopaedic Nurses Examination in Cape Town.

NEW ZEALAND

NEW ZEALAND ORTHOPAEDIC ASSOCIATION

ANNUAL MEETING 1951

The annual meeting of the New Zealand Orthopaedic Association was held at the Wellington Hospital on June 15 and 16, 1951. The Association had the pleasure of entertaining at the meeting Sir Reginald Watson-Jones, who was present in New Zealand in the capacity of Sims Travelling Commonwealth Professor. Sir Reginald delivered an opening address and led the discussion on the papers and cases that were presented.

Volkmann's ischaemic contracture—*Mr N. W. Nisbet* (Dunedin) reported a case treated by a muscle slide operation. Marked improvement in function was secured.

Bone lengthening—*H. Walden Fitzgerald* (Dunedin) described the case of a high country sheep farmer aged thirty with lower limb shortening of one and three-quarter inches due to an old fracture of the tibia and fibula. He was intolerant of a surgical boot and had a painful and stiff lumbar spine which rendered a compensatory pelvic tilt difficult. The patient refused leg shortening. Leg lengthening was carried out by an oblique tibial osteotomy and gradual pin distraction by means of an external apparatus with turn-buckle arrangements. Distraction took place daily and at the end of two weeks an increase in length of one and a half inches had been gained. The tendo calcaneus was not divided. Slight posterior angulation occurred and was corrected. A plaster incorporating the pins was subsequently applied; and later a caliper was fitted. The patient returned to work seven months after operation. The difficulties and dangers of bone lengthening were discussed.

Fractures of the lower end of the humerus in children—*Mr H. Kenrick Christie* (Wanganui) advocated the use of continuous traction by a Kirschner wire through the olecranon in the more difficult and unstable supracondylar fractures of childhood. He showed a frame attached to the bed by means of which traction was maintained. The method had been used in fifteen cases and detailed results were given. *Sir Reginald Watson-Jones* complimented Mr Christie on his results. He stated that good results could be obtained in two ways—the easy way and the hard way and he thought that Mr Christie had chosen the hard way. Considerable discussion followed. The consensus was that in childhood open operation and pin traction were almost never required. Equally injurious was repeated manipulation. Good alignment afforded a good result even in the absence of perfect anatomical reduction.

Adduction fractures of the ankle in childhood—*Mr William Parke* (Gisborne) adopted the classification of Aitken of first degree fractures—a simple crack in the lower tibial epiphysis without displacement—and second degree fractures, in which the fracture line extended upwards from the medial angle of the ankle mortise across the epiphysial plate, with separation of a fragment of metaphysial bone and upward displacement of the medial malleolar fragment. Traumatic epiphysial arrest in second degree fractures was discussed.

An illustrative case showed medial tilting of the tibial articular surface due to premature fusion of the medial aspect of the epiphysial plate and varus deformity of the foot. Osteotomy of the tibia from the medial aspect was advocated. The osteotomy was opened up on the medial side and packed with chips from the divided fibula. This helped to reduce shortening. Without interference the varus deformity is progressive until epiphysial closure occurs. If the deformity commences in early childhood repeated operation may be necessary. Epiphysiodesis of both bones at the ankle may prevent this at the expense of much shortening. The etiology of this traumatic epiphysial arrest has been accepted as crushing of the medial portion of the plate. The speaker cast doubts on this view. He considered that upward displacement of the malleolar fragment caused malalignment of the epiphysial plate so that union of the cancellous bone of the epiphysis and metaphysis could occur at the fracture site with production of a bony lock and inevitable premature epiphysial arrest. It should be possible to prevent this by realigning the epiphysial plate shortly after injury. An illustrative case was described in which open reduction was performed with internal fixation with one stainless steel screw. Six months after injury the epiphysial plate was clearly defined and there was no evidence of epiphysial arrest.

Rupture of the brachial artery complicating dislocation of the shoulder—*Mr Selwyn Morris* (Auckland) reported a case.

Resection of the fibula for osteomyelitis—*J. Leslie Will* (Christchurch) described a case in which resection of the shaft of the fibula for osteomyelitis was followed by regeneration, fracture and bony union.

Clinical cases—Among the interesting clinical cases shown were: calcinosis universalis (*Mr Jennings*); insidious onset of bilateral bony ankylosis of the hips in a young man with no changes in sacro-iliac joints or spine, thought to be a low grade infective arthritis (*Mr Jennings*); a successful result following quadriceps-plasty in a stiff knee due to old osteomyelitis of the femur (*Mr J. K. Cunninghame*); aneurysm of the femoral artery from artificial limb (*Mr Kennedy Elliott*); electrolytic destruction of the lower two-thirds of the radius in a fracture plated in 1912 which simulated osteoclastoma (*Mr Kennedy Elliott*); a series of cases to illustrate various aspects of carpal tunnel compression of the median nerve (*Mr Kennedy Elliott*); a series of cases to illustrate the results of Smith-Petersen cup arthroplasty, one patient giving a convincing exhibition of jumping (*Mr Alexander Gillies*).

Honorary Fellowship—At the annual meeting Sir Reginald Watson-Jones was elected an Honorary Fellow of the New Zealand Orthopaedic Association.

AUSTRIA

AUSTRIAN ORTHOPAEDIC ASSOCIATION

1950 MEETING

A meeting of the Austrian Orthopaedic Association was held in December 1950, under the presidency of Professor Erlacher.

Arthrosis deformans—*Mr A. Hartwich* stressed the importance of the problems caused by arthrosis deformans in connection with the increasing average age of the population. He suggested general outlines of treatment and pleaded for special measures by public health and social welfare institutions. In *discussion*, speakers gave their views on various conservative and operative methods and their results.

1951 MEETINGS

A meeting was held in February 1951. Papers were read. A *Festival meeting* was held from May 15 to 17, 1951, for the twenty-fifth foundation of the Unfallkrankenhaus in Vienna (Professor Dr L. Boehler) from the Austrian Traumatological Section. The main subject for discussion was fracture of the neck of the femur, and especially the causes of necrosis of the head of the femur after this injury.

ITALY

ITALIAN SOCIETY OF ORTHOPAEDIC AND TRAUMATIC SURGERY

The thirty-sixth annual meeting of the Italian Society of Orthopaedic and Traumatic Surgery was held in Rome on October 20 to 22, 1951, at the Orthopaedic Hospital of the University, under the presidency of Professor C. Marino-Zuco. The principal subject discussed was osteosynthesis in modern traumatology. An official report on this subject was presented by Professor C. Marino-Zuco and his co-workers, Drs N. Pancino, V. Pietrogrande, A. Rampoldi, Mr Ascoli, E. Tranquilli-Leali. On this same subject eighty-four papers were presented and twenty-three further papers were read on different subjects. The meeting was attended by many eminent orthopaedic surgeons from other countries, including Professor R. Merle D'Aubigné and Dr P. Truchet (France), Professor G. Hohmann, Professor C. Mau, Professor M. Lange and Professor K. Niederecker (Germany), Professor Garaizabal-Bastos and Dr Vaquero (Spain), and Professor A de Araujo (Brazil).

The thirty-seventh annual meeting of the Society will be held in Genoa in October 1952, when the principal subject to be discussed will be the surgical treatment of the paralytic shoulder.

GERMANY

GERMAN SOCIETY OF ORTHOPAEDICS

The thirty-ninth Congress of the German Orthopaedic Society was held in Heidelberg on September 5 to 8, 1951, under the presidencies of Professor Weil (Heidelberg) and Professor Lindemann (Hanover). The secretaries to the conference were Professor Lange (Tölz), Professor Mau (Hamburg) and Professor Hackenbroch (Cologne). The first day of the meeting included a visit to the orthopaedic clinic of Heidelberg University. Papers were read on the three days following the opening day and the main subject of discussion was that of arthroplasty. Among those taking part in the discussions were Professor Beck (Berlin), Professor Chapchal (Utrecht), Professor Chiari (Vienna), Dr Judet (Paris) and Professor D'Aubigné (Paris).

We have been informed that Sir Harry Platt (Manchester) has been elected an honorary member of the German Orthopaedic Society.

CORRESPONDENCE

INTRAMEDULLARY NAILING OF FRACTURES

From Mr John Charnley, Manchester, England

To the Editor,
Journal of Bone and Joint Surgery.

Dear Sir,

During a recent bone-grafting operation for non-union of the femoral shaft a way of using the Küntscher nail occurred to me of which I had not previously heard, and which seems worthy of being noticed by all who may be faced with the same situation.

In long-standing non-union of the shaft of the femur the medullary canal becomes so wide, as a result of osteoporosis, that the thickest Küntscher nail is too small and the

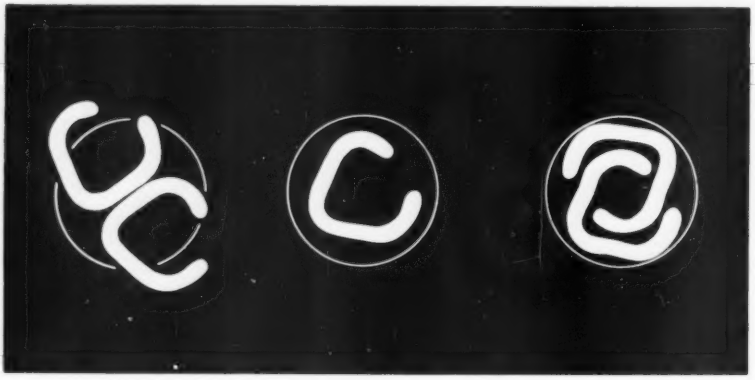

Fig. 1

osteosynthesis is therefore unstable against rotatory strains. With Küntscher nails of the clover-leaf pattern it is possible to interlock two nails inside each other and use the combination as a single nail. This interlocking is secured by sliding one nail into the other so that the two limbs of one enclose one limb of the other. The result is that the outside circumference is greater than one nail alone, but less than that of two nails placed side by side (Fig. 1). By mating nails of different diameters a combination can be found which should fit snugly into the widest medullary canal.

This useful trick would seem to be another reason to support the superiority of the clover-leaf pattern over the solid type of nail. Removal of the two nails might be a little difficult as full penetration of the extractor through the "eyes" of the nails is obstructed. If one nail is left projecting one or two centimetres more than the other this difficulty would not exist.

Yours truly,

October 5, 1951. JOHN CHARNLEY.

TUBERCULOSIS OF THE SPINE

From Mr Robert Roaf, Liverpool, England

To the Editor,
Journal of Bone and Joint Surgery.

Dear Sir,

We are indebted to Dr Dobson for a comprehensive analysis of the results we can expect from the best conservative treatment of tuberculosis of the spine. It is because Dr Dobson's figures and opinions will carry great weight throughout the world that I venture to indicate a number of points on which one could legitimately disagree with his conclusions.

Firstly, he does not stress sufficiently the serious nature of spinal caries. In his summary he says that the mortality was 18·7 per cent, but this is the mortality during treatment. At the three-year follow-up the mortality was at least 27 per cent (155 out of 569—assuming the 103 cases untraced were unselected) and possibly 38 per cent (256 out of 672 if all the untraced patients were dead). These figures include children and adults; there are reasons for believing that the five-year mortality in adults is higher still, especially in thoracic lesions.

I also feel that Dr Dobson underestimates the evil effects of abscess formation—surely the mediastinal abscess is a frequent cause of both reactivation and of spread to other vertebrae, and it should be treated strenuously by open drainage and removal of necrotic tissue; aspiration alone is inadequate. The presence of an abscess is one important cause of failure of spinal-fusion operations; either fusion fails to occur or the disease recurs at the same or a different level. This is important in the prevention of late onset paraplegia, and I am sorry that Dr Dobson does not differentiate early and late onset paraplegias as the two types differ in so many important respects. Late onset paraplegia is unlikely to occur if there is sound healing, bony fusion and slight deformity; a well-timed spinal graft will usually lead to this desirable condition. In failing to consider both the timing of the fusion operation and the presence or absence of an abscess, I feel that Dr Dobson has hardly been fair to operative treatment.

My reason for writing this letter is not to criticise or belittle Dr Dobson's work, for which I have a great respect. It is because I fear that the weight of his authority may lead orthopaedic surgeons to accept his opinions both on prognosis and treatment as fixed and permanent, and they may fail to recognise that operative interference has a place as an aid to conservative treatment in cases of tuberculosis of the spine.

Yours truly,

November 19, 1951. ROBERT ROAF.

Book Reviews

INSTRUMENTS AND APPARATUS IN ORTHOPAEDIC SURGERY. By E. J. Nangle, M.B., F.R.C.S. Formerly Resident Surgical Officer, Royal National Orthopaedic Hospital, Stanmore; and R.S.O., Wingfield-Morris Orthopaedic Hospital, Oxford. Beit Fellow. 10 × 7½ in. Pp. xv + 231, with 200 figures. Index. 1951. Oxford: Blackwell Scientific Publications. Price 42s.

I have nothing but praise for this book, which is almost the first of its kind in order of appearance and certainly the first in quality. The need for it is particularly strong at present, when the art of conservative orthopaedics is being pushed into the background by another surgical revolution, and it answers that need well. It will give welcome help to any orthopaedic surgeon, however senior, and it should be invaluable to orthopaedic ward-sisters and to residents. The title is a little misleading if by "instruments" one means the tools of the operating theatre. Perhaps the word "appliances" might have been better. Neither should the title be taken to indicate that this is a complete encyclopaedia, for it is not. Mr Nangle has very wisely restricted his field to the appliances and apparatus in use at the two orthopaedic hospitals where he has done most of his work, the Wingfield-Morris and the Royal National. It therefore follows that there are no gaps of any note in the subject matter, and, though it is not claimed that every appliance one can meet is described in this book, it would be quite true to claim that at least one way of doing everything is described in enough detail to be really helpful.

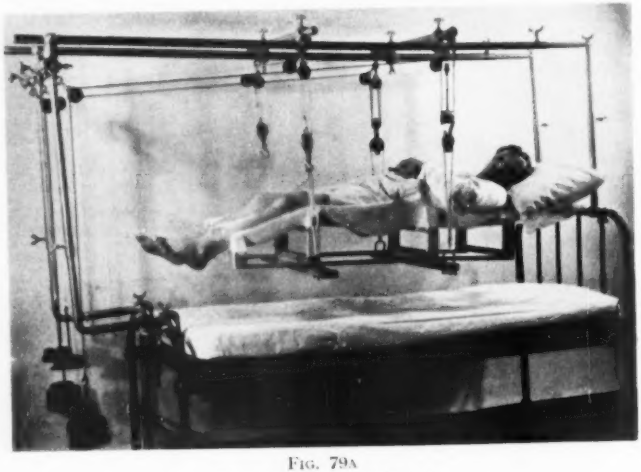

FIG. 79A

Plaster bed counter weighted.

The book starts by laying down basic principles of splintage, and very wisely devotes a whole chapter to the application of these principles in poliomyelitis. The appliances themselves are described with manufacturing details, as if this were the orthopaedic pharmacopoeia, which indeed it is. There is an excellent chapter on mechanical respirators, and one is very pleased to see Mr Nangle give due prominence to the suspension and counterpoising devices which he and his colleague G. F. Domisse first described some few years ago (British Journal of Surgery, **1947, 34,** 395). A chapter by J. T. Scales on plastics and their preparation and use in orthopaedics outlines what may be the shapes of things to come, and the whole book is beautifully produced.

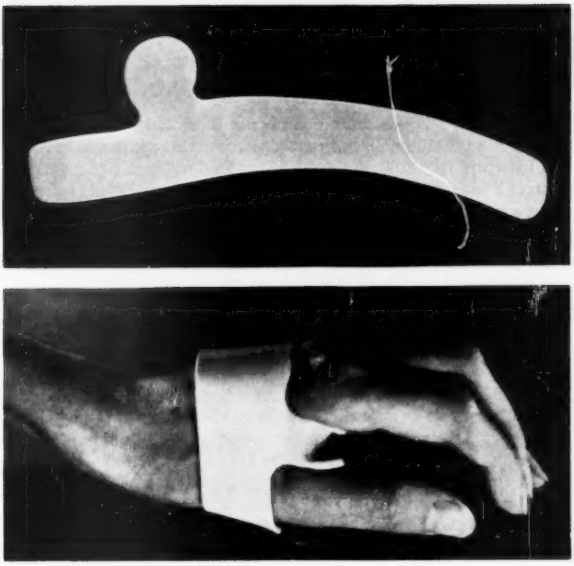

FIG. 96
The Cholmeley opponens splint, applied for thenar muscle paralysis from poliomyelitis.

The illustrations really illustrate the points in question, and must have demanded a great deal of care and trouble and collaboration between the author and the photographer. Despite this, the book is by no means expensive at to-day's prices. It is seldom that a reviewer is able to give himself the pleasure of enthusiastic praise, but this book is as pleasant to review as it is to study.—D. Ll. GRIFFITHS.

THE AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS INSTRUCTIONAL COURSE LECTURES. Editor, Charles N. PEASE, M.D., and Sam W. BANKS, M.D. 11 × 9 in. Volume 7, 1950—Pp. viii + 285, with numerous figures. Volume 8, 1951—Pp. viii + 356, with numerous figures. Indices. Ann Arbor, Michigan: J. W. Edwards Bros. Inc. Price, Volume 7—$6.50; Volume 8—$8.50.

The American Academy of Orthopaedic Surgeons arranges an instructional course every year in connection with its annual congress and each of these volumes contains a selection of the lectures delivered at one course. The contributions are of the highest standard and really amount to the carefully prepared presentation by leading authorities of an up-to-date survey of a wide variety of subjects. The lectures are of such uniform excellence, and there is so little personal axe-grinding, that it is unfortunate they are hardly known in the United Kingdom. It is invidious to pick out only a few names, but this is necessary to give an idea of the field covered. The thirty-three lectures in volume 7 include contributions by Copeland on cartilaginous tumours, Snapper on metabolic diseases, Strandskov on human genetics, Steindler on idiopathic scoliosis, and Bechtol on muscle physiology. Volume 8 contains forty-six lectures, including reports by sixteen surgeons on the work organised by Hugh Smith and carried out in conjunction with the Department of the Army and the National Research Council on medullary fixation of the femur, lectures by Kendall and by Hench on cortisone, by Colonna on congenital dislocation of the hip, and by Bosworth on slipping of the upper femoral epiphysis.

Dr Pease and his assistants must be congratulated—perhaps it would be better to say thanked—for the skill with which they have carried out their work. This series should be owned and studied by every orthopaedic trainee, and also by every surgeon who wishes to keep abreast of orthopaedic practice in the U.S.A.—Philip WILES.

EVALUATION OF DISABILITY AND REHABILITATION PROCEDURES OF PATIENTS WITH SPINAL CORD LESIONS. By George G. Deaver, M.D., Clinical Professor of Rehabilitation and Physical Medicine, New York University College of Medicine. 10¼ × 7 in. Pp. 62. 1947. New York: Institute for the Crippled and Disabled. Paper cover. Price $1.

The title of this monograph is misleading. It is not a critical analysis of the value of various procedures employed in the rehabilitation of paraplegic patients: it merely gives an outline of the methods used at the Institute for the Crippled and Disabled with special details of active exercises to develop arm and trunk muscles. In general, the treatment advocated is sound and is much the same as is used at any other paraplegic centre but it is difficult to see for whom the volume is intended. There is not enough in it to be of real help to anyone who works at a paraplegic centre, and yet there is too much detail for the general reader; nor is it suitable for the patient or his relatives. Unfortunately, there is no reference to the early treatment of paraplegic patients, so that the book is not even of value as a reference volume in the accident ward of a general hospital.—Robert Roaf.

ORTHOPAEDIC NURSING. By Mary Powell, S.R.N., M.R.S.P., Orthopaedic Nursing Certificate. Assistant Matron in charge of Children's Annexe, and Sister Tutor in Orthopaedic Nursing, Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry. 8½ × 6 in. Pp. xii + 400, with 210 figures. Index. 1951. Edinburgh: E. & S. Livingstone Ltd. Price 25s.

This interesting book is designed, as is stated in the preface, primarily for nurses and physiotherapists in orthopaedic hospitals. As Miss Powell is an After-care Sister there is an understandable emphasis on the after-care and physiotherapy side of orthopaedic nursing. It is assumed that the nurse is familiar with elementary nursing procedures such as barrier nursing and the importance of the prevention of the spread of infection: in the chapter on poliomyelitis, for example, the subject of isolation treatment in the acute stage is dealt with in four lines. In several places greater detail of actual nursing procedures would perhaps have been an advantage, particularly the nursing of a patient in a respirator. Much information is given in relation to splints and appliances—their measurements, application, and maintenance being set out in detail. The chapters on tuberculosis of bone and joint present a clear and complete description of the methods of treatment generally adopted in orthopaedic hospitals in this country. The section on congenital deformities is particularly well illustrated, showing, for example, methods of reduction of congenital dislocation of the hip on a double abduction frame. The Batchelor plaster is mentioned as one method of maintaining reduction: a little more indication of some of the difficulties which arise in nursing a child in a Batchelor plaster might have been valuable. Chapters 24 to 28 deal with the treatment of fractures and other injuries. This section will be of particular value to the nurses whose work is carried out in a hospital dealing largely with long-term orthopaedic cases. The author is to be congratulated on providing a text-book that is perhaps the best that has been written on the care of the orthopaedic patient. It is no serious criticism, but an indication of the scope of the book, to suggest that an equally good title might have been "Orthopaedics for Nurses." There are in all some two hundred illustrations and the book can be recommended as a very worth while addition to training school libraries.—Elizabeth M. Stone.

CHIROPODIAL ORTHOPAEDICS. By Franklin Charlesworth, F.Ch.S., Consulting Chiropodist, Hope Hospital, Salford; School Health Service, Salford; Department of Civic Welfare, Salford. Foreword by Sir Harry Platt, M.S., F.R.C.S., F.A.C.S. 8½ × 5½ in. Pp. viii + 255, with 131 figures. Index. 1951. Edinburgh: E. & S. Livingstone Ltd. Price 25s.

I advise potential readers not to be put off by the title of this book. The juice inside a peach is more attractive than the skin. There are innumerable tips for making patients more comfortable. It is probable that if the Editorial Board of this Journal had had to "vet" it, the book would have been pruned somewhat. But presumably publishers of this calibre must have some space on which to spread their art. They spread it very pleasantly. It seems to me that this book should be regarded as an addendum to the book by Mary Powell on Orthopaedic Nursing.—Bryan McFarland.

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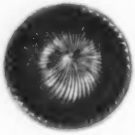

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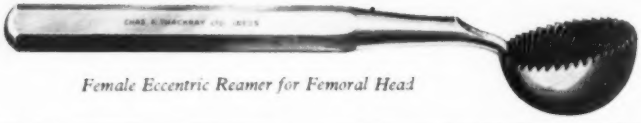

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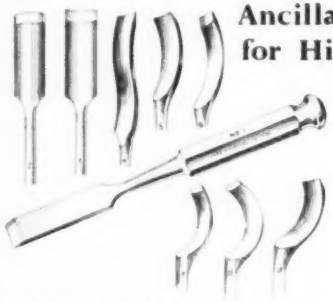

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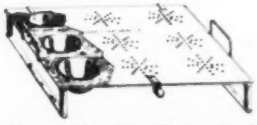

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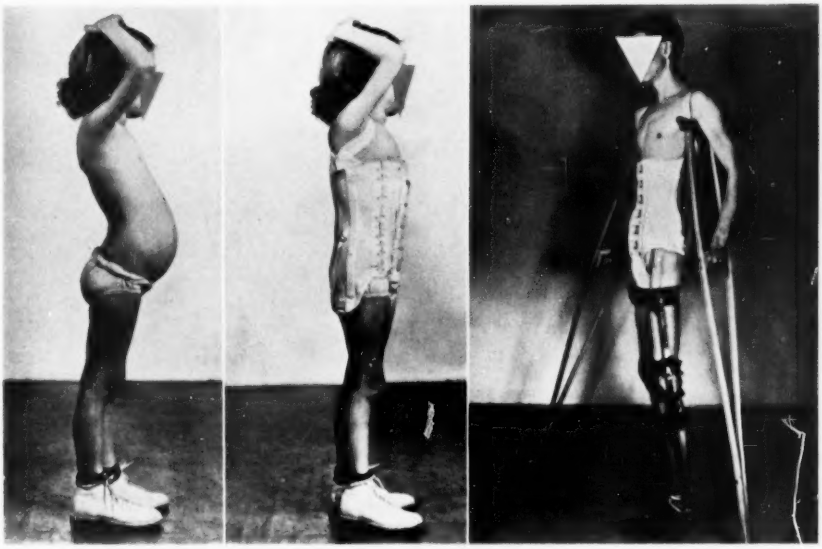

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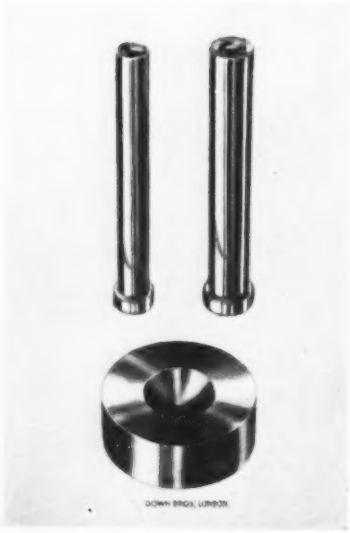

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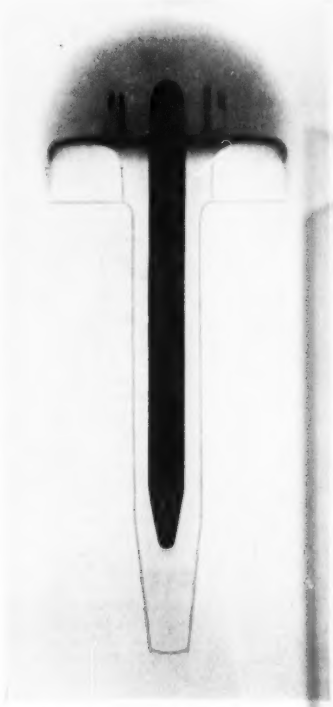

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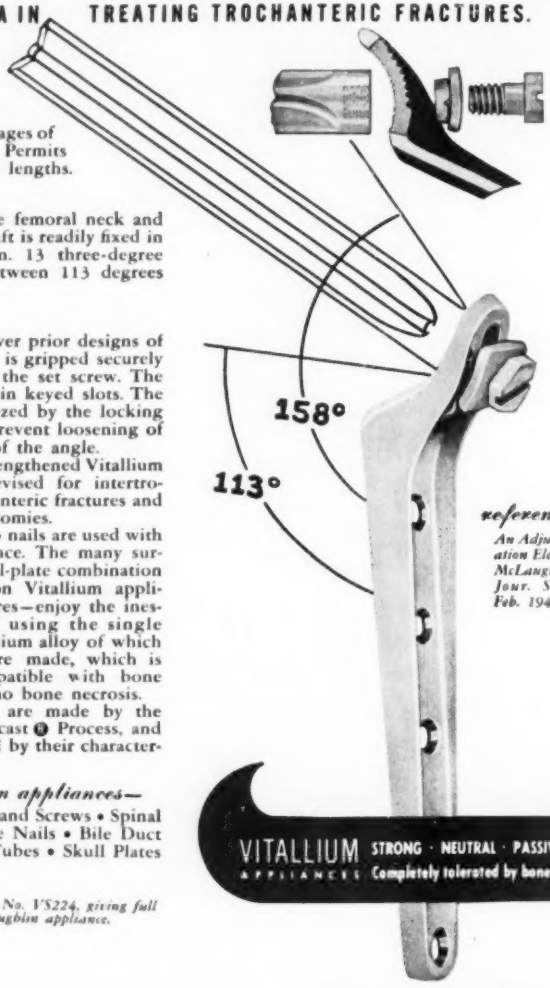

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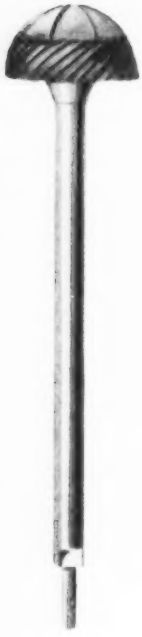

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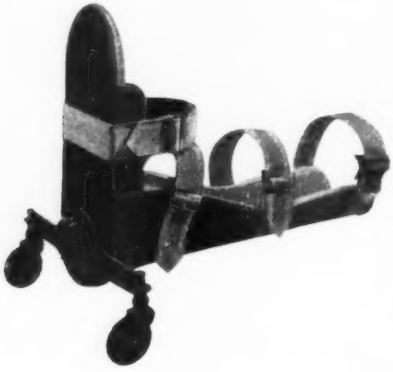

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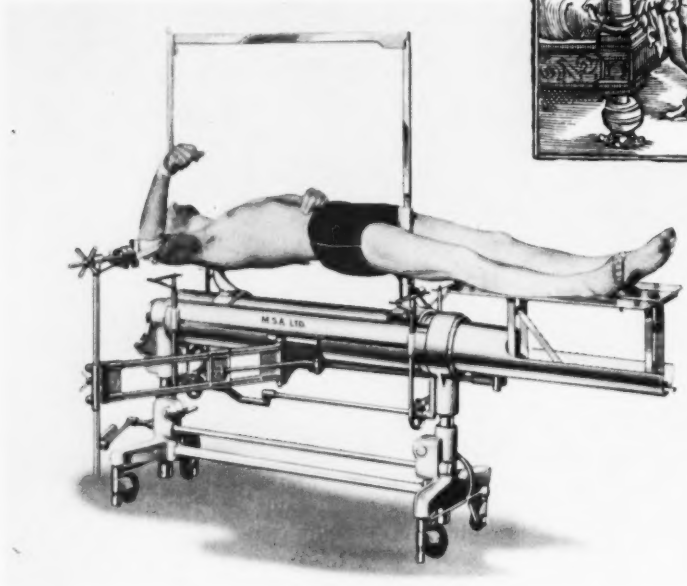

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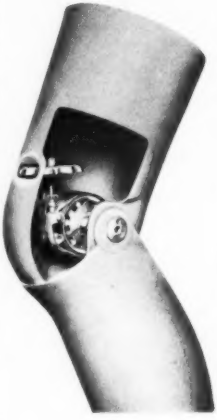

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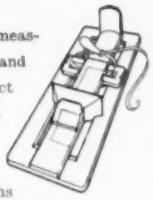

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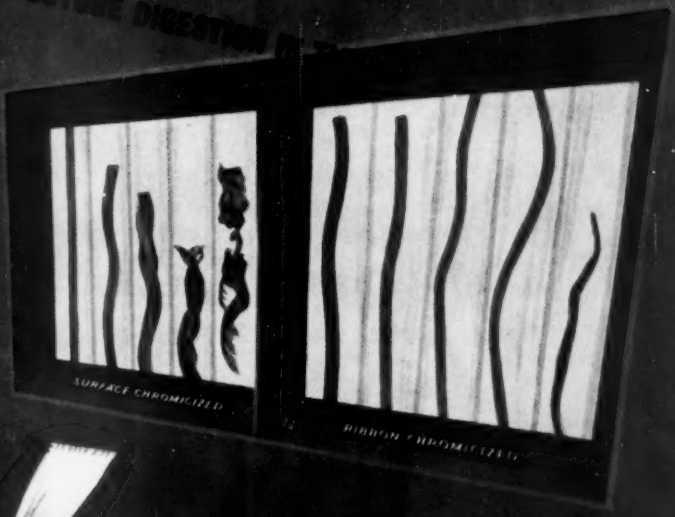

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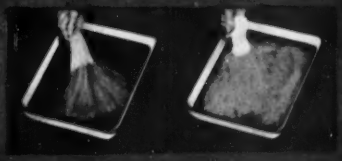

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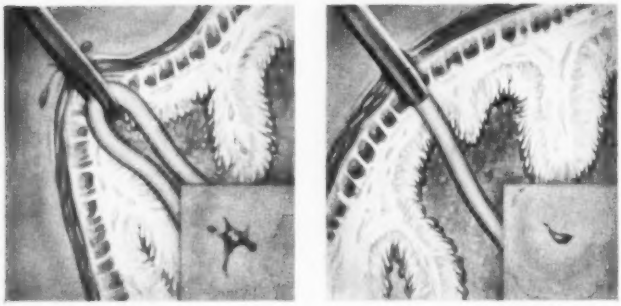

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